The Journal

of the

Louisiana State Medical Society

$4.00 Per Annum, 35c Per Copy
Vol. 108, No. 11

NOVEMBER, 1956

Published Monthly
1430 Tulane Avenue, New Orleans 12, La.

ABDOMINAL WOUND COMPLICATIONS IN OBSTETRIC AND GYNECOLOGIC SURGERY *

FRANK G. NIX, M. D.
GEORGE F. SUSTENDAL, M. D.
CONRAD G. COLLINS, M. D.
FREDERICK R. BRADEN, M. D.
NEW ORLEANS

Despite adherence to the precepts of Halstead, the advent of better detergents, antiseptics, blood transfusion, vitamins, amino acids for intravenous use, and antibiotics, wound complications continue to occur. It is our feeling that if wounds in which suspected hematoma, abscess, seroma, dehiscence, or evisceration, alone or in combination, were explored in the operating room rather than in the patient's room or ward, the more serious early or late complications might be reduced or avoided.

Massive evisceration with large amounts of protruding omentum and/or bowel with a long time interval between its occurence and repair certainly is not conducive to lower mortality. Furthermore, disruption of the wound involving the fasciae layers, even though the peritoneum is intact is a fertile field for the development of post-operative incisional hernias. Obstetricians have learned the value of not examining a patient with suspected placenta praevia anywhere except in the delivery room or surgical amphitheatre, where all available facilities for the control of hemorrhage and the performance of any operative procedure are immediately available. The same attitude regarding wound complications has been adopted by the Tulane Gynecological Service at Charity Hospital in New Orleans. If a wound complication is suspected, removal of sutures, probing of the wound in the patient's room, or a "watch and wait" attitude are not utilized; the patient is removed to the operating room, which has been set up for laporatomy. Here the sutures are removed from the skin and the wound explored. Anesthesia is administered if necessary. Abscesses, seromas, and hematomas are thoroughly evacuated. The integrity of the fascial suture line is determined. If the latter shows any evidence of disruption in continuity, the sutures in the fascial layer are removed, and the line of peritoneal sutures explored. Any collection of blood, pus, or serum is evacuated and active bleeding points ligated. Defects in the peritoneum and/or fascia are repaired. This policy was begun in June, 1946, in one ward of the Tulane Unit and since January, 1949, it has been in effect on all the gynecologic and obstetric wards assigned to the Tulane Unit. Our experiences and results with this procedure form the basis of this communication.

MATERIAL

The material of this study was collected at Charity Hospital during the ten years from January, 1946, through December,

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* Presented at the Seventy-sixth Annual Meeting of the Louisiana State Medical Society, Alexandria, La., April 24, 1956.

From The Dept. of Obstetrics and Gynecology, Tulane University and Charity Hospital, New Orleans, La.

1955, and includes 5200 consecutive gynecologic laporatomies. These operations cover the full scope of gynecologic abdominal surgery. The material consists of 140 cases of wound explorations with subsequent secondary closure in which the mortality rate was 5.7 per cent. The complications range from superficial abscesses and hematomas through complete evisceration. There were 31 total eviscerations in this series with an incidence of 0.6 per cent and a mortality of 19 per cent; 20 of these were discovered at the time of exploration, a discovery rate of 64.5 per cent. Also discovered during the course of exploration were 40 wound ruptures that extended to the peritoneum exclusive of complete evisceration, a discovery rate of 37 per cent. There were 8 deaths with an uncorrected mortality of 5.7 per cent, considerably under the national average. This death rate could be further corrected.

The indications for wound exploration with their corresponding frequency of occurrence are listed in Table 1. Exploration findings and degree of involvement are tabulated against the pre-exploration diagnosis. (Table 1)

GENERAL FACTORS

Certain general factors have been given extensive consideration in all studies of wound disruption. These are discussed below.

Age: In the general literature an average age of 44 years is reported for wound disruption. In our series the average age was 43, with the youngest being 17 years and the oldest patient 86 years. The patients were grouped more or less according to the usual age for gynecologic surgery. (Table 2.)

TABLE 2
INCIDENCE OF WOUND DISRUPTION BY AGE GROUPS

Age (years)	Number of wound complications	Number of eviscerations
10 - 19	3	0
20 - 29	17	3
30 - 39	33	11
40 - 49	66	10
50 - 59	12	4
60 - 69	4	2
70 - 79	3	1
80 - 89	2	0

Malnutrition: Lack of proper nutrition with consequent anemia, low serum protein, and avitaminosis have been discussed as contributing factors to wound complications.[1] Although all patients in this series were of a clinical level with the exception of those cases done as emergency, an attempt was made to stabilize the nutrition of all patients. This was revealed by the fact that in 140 cases with complications the hematocrit was normal in 48 per cent, high in 16 per cent and low in 36 per cent. In the 118 patients in whom plasma protein levels were recorded, 30 per cent had high levels, 18 per cent were low, and 52 per cent were normal; 80 per cent of these patients received intravenous vitamin C at the time of the initial operation.

Malignancy: 112 cases or 80 per cent of the patients with wound disruption were operated upon for benign lesions; 28 cases or 20 per cent had proven malignancies; of the 31 cases that eviscerated 22 of the cases or 72 per cent were in the former group and the remaining 9 cases or 30 per cent had been operated upon for a malignant lesion.

Anesthesia: Of the 140 patients with wound disruption 56 per cent received

TABLE 1
PRE-EXPLORATION DIAGNOSIS, EXPLORATION FINDINGS, AND DEGREE OF INVOLVEMENT IN CASES OF WOUND COMPLICATIONS

Diagnosis prior to exploration	No.	%	Exploration			Involvement		
			Hem.	Abs.	Ser.	Fascia	Periton.	Total
Hematoma	64	46.4	59	4	2	40	25	0
Abscess	31	21.4	0	31	0	18	9	3
Seroma	7	5.0	4	0	3	4	3	0
Tenderness	5	3.6	5	0	0	4	1	0
Evisceration	28	20.0	3	5	14	0	0	27
Bleeding	5	3.6	4	0	0	2	2	1

spinal anesthesia at their initial surgery; 39 per cent received inhalation anesthesia, and the remaining 5 per cent were performed under local infiltration. Spinal anesthesia, when not contraindicated, is the anesthetic of choice on the Tulane service.

Incision: The type of incision has been credited with a major role in wound disruptions. Some authors [2, 3] state that anatomic incisions, such as the transverse, are superior to longitudinal incisions. In any study that includes only gynecologic cases the longitudinal incision, principally the subumbilical midline is bound to predominate. That is true in this study. In 89.4 per cent of these cases the incision was located in the midline; 7.1 per cent were of of the paramedian type; and in 3.5 per cent a transverse incision was used.

Closure: The type of closure in wound disruption has always been a matter of dispute.[4] All of these cases were closed in anatomic layers. The suture materials used are indicated in Table 3. Retention sutures were not used. The vast majority of the 5200 cases were closed with interrupted cotton sutures. (Table 3).

TABLE 3
INCIDENCE OF WOUND DISRUPTION BY TYPE OF CLOSURE

Periton.	Fascia	Skin	Incidence of complications %	Incidence of evisceration %
G	C	C	65	56
C	C	C	14	28
G	G	C	14.4	12
G	G	S	2.6	4
G	S	S	2.6	0
Figure of 8			1.4	0

G—catgut C—cotton S—silk

Race: 79 per cent of our wound complications occurred in individuals of the colored race. While it is true that our negro gynecologic service at Charity Hospital is somewhat larger than the white service, the difference is not enough to account for this predominance. It was felt that the presence of many of these complications was due to deficient personal hygeine. Initially it was the practice on this Service to do vaginal and abdominal preparations, employing a through scrubbing with tincture of green soap followed by ether and merthiolate. For the last few years, only a thorough scrubbing with phisohex, has been employed. Despite this fact, approximately 25 per cent of our wound complications were due to infection.

POSTOPERATIVE FACTORS

Increased intra-abdominal pressure is always discussed as a major cause of wound disruption.[5] In our series, however, only 46 patients, or 33 per cent gave any indication of abnormal distention. It has been and is a policy of the service to introduce nasogastric suction at the first sign of distention. Thus it is possible that early distention may not have been recorded in some of these patients. Eighteen per cent had increased intra-abdominal pressure due to coughing; 14 patients or 10 per cent had their postoperative course complicated by vomiting. In only 1 patient was sneezing noted to be the cause of evisceration.

DIAGNOSIS

The diagnosis in most of these cases was made at the operating table; this was our intention. Any wound that showed unusual fluctuance or induration, the presence of discharge (purulent, bloody, or serosanguinous), or unusual tenderness were all explored. The average time of wound exploration was the seventh day, or about the time that the sutures would ordinarily be removed. However, 5 cases were explored on the first postoperative day in the presence of active bleeding, and 2 cases were explored as late as the thirtieth day in the presence of wound infection. The greatest spread of explorations occurred between the sixth and tenth day. These wounds, with the exception of wound infections which were packed open and resutured at a later date, were all closed for the second time at the time of exploration. In almost all cases through and through sutures were used, either in the form of a mattress or figure of eight suture; nonabsorbable materials were used in all cases. The vast majority of these patients were closed with crochet cotton. In no instance was a patient so sick as to be unable to stand secondary

closure. In only 3 cases was tertiary closure necessary. (Table 4)

TABLE 4
MORTALITY IN WOUND DISRUPTION

Year	Evisceration	Deaths
1946 - 47	2	0
1947 - 48	2	1
1948 - 49	5	0
1949 - 50	5	1
1950 - 51	8	2
1951 - 52	5	3
1952 - 53	1	0
1953 - 54	1	0
1954 - 55	2	1

CASE REPORTS

Case No. 1.—E.T. a 38 year old colored female who had a caesarian section at six months gestation when almost moribund from porphyria. She eviscerated on the seventh day after suture removal and died on the eighth. The final diagnosis was porphyria with evisceration.

Case No. 2.—E.H. a 56 year old colored female with a diagnosis of adenocarcinoma of fundus. At operation she was found to have generalized carcinomatosis. A hysterectomy was done, the patient's sutures were removed on the tenth postoperative day and evisceration occurred. She died on the twenty-seventh postoperative day with a diagnosis of generalized carcinomatosis with evisceration.

Case No. 3.—H.B. a 48 year old colored female who had a total abdominal hysterectomy and bilateral salpingo-oophorectomy for multiple fibroids. Her sutures were removed on the eighth postoperative day and her wound was explored on the sixteenth postoperative day. A wound abscess was found extending to the peritoneum. She died on the twenty-fifth postoperative day of a brain abscess and had a final diagnosis of brain abscess with thrombocytopenic purpura.

Case No. 4.—C.R. a 33 year old colored female who had a caesarian section (elective repeat section). Her wound was explored on the first postoperative day because of active bleeding. She died on the ninth postoperative day from lower nephron nephrosis as a result of an incompatible blood transfusion.

Case No. 5.—B.K. a 20 year old colored female who had a caesarian section with a diagnosis of eclampsia and abruptio placenta. Her wound was explored on the eleventh postoperative day and a hematoma was found extending to the peritoneum. This was evacuated and the wound closed. She died on the twenty-ninth post-operative day in convulsions. A cerebral thrombosis was found.

Case No. 6.—F.L. a 33 year old colored female who had a pelvic exenteration for recurrent cancer of the cervix. Sutures removed on the tenth postoperative day. Wound explored on the tenth day and an abscess was found extending into the pelvis, the abscess was drained and the wound closed secondarily with drainage. She expired on the eighteenth postoperative day with diagnosis of peritonitis, pyelonephritis and evisceration.

Case No. 7.—P.J. a 37 year old colored female who had had a total abdominal hysterectomy and bilateral salpingo-oophorectomy for Grade IV cancer of cervix, done in another hospital. She was admitted for recurrent cancer of cervix. At laparotomy, the recurrence was found to be inoperable. Sutures were removed an the eighth postoperative day. Complete wound dehiscence occurred on the ninth postoperative day. At exploration an abscess was found extending into the peritoneal cavity. Secondary closure with drainage was done and the patient was discharged on the twenty-eighth postoperative day. She died two months later at home from metastatic carcinoma.

Case No. 8.—W.McC. a 65 year old white female admitted for recurrent cancer of the cervix following irradiation. Sutures removed on the seventh day and wound inspected. Hematoma extending to peritoneum found and secondary closure done. Developed bilateral ureteral obstruction on ninteenth day, and in spite of catheter drainage patient died of overwhelming sepsis. Final diagnosis was necrotizing papilitis and severe pyelonephritis.

DISCUSSION

A comparison of the results of our work and those of other authors may be obtained by referring to Table 5.[6-13] It is apparent from this table that our mortality at the time of this publication is considerably lower than that existing under other forms of treatment.

Mayo and Lee with a mortality of 18.1 per cent compared to our mortality rate of 19 per cent have published the lowest figures to date. Other mortality figures varied from 30 to 50 per cent.

The apparent effectiveness of this form of treatment may be somewhat enhanced by the fact that all of our incisions were of the lower abdominal type and in most cases midline. This type of incision is less prone to wound rupture than those involving the upper abdomen. Another factor that may have influenced our figures is the fact that wound rupture is more apt to occur in the male than in the female. However, our percentage of malignancies in this series is somewhat higher than the

TABLE 5
RESULTS OF MANAGEMENT OF ABDOMINAL WOUND COMPLICATIONS BY SEVERAL AUTHORS

Author	No. Cases	Ave. Age	% Female	% Malig.	% Ob. & Gyn.	% Mortality	% Infect.	% Lower Abdom. Incision
Charity Hospital	140	43	100	20	100	19	25	100
Fallis	50	45	45	16.3	28.6	34	20	47
Glenn et al.	22	50	9	27	0	45.5	27.2	32
Hinton	19	42	30	10.5	0	21	16	5
Singleton	61	36	73	6.5	65.5	31	60.7	85
Totten	47	45	20	15	8.5	40	36	49
Mayo et al.	94	54	11.7	39.4	0	18.1	0	25.5
Bowen	34	45	41	35.5	17.6	50	12	44

average and would favor a higher percentage of wound ruptures and an increase in mortality because of the accompanying debility.

CONCLUSION

It is our feeling that the routine exploration of all suspected wound complications is a worthwhile procedure. This is supported by the fact that of 31 eviscerations 64.5 per cent were discovered before the actual occurrence of extrusion of abdominal contents through the skin wound, making it possible to handle these cases under sterile technique in the operating room. Of 109 other complicated cases, 36.7 per cent were found to have involvement extending to the peritoneum which would invariably result in postoperative hernia. It is our opinion that in no other way than by proper exploration of these wounds would it have been possible to discover many of these cases and prevent future surgery. We firmly believe that this method of management of wound complications has considerably reduced our death rate from evisceration.

REFERENCES

1. Krayhill, W. G.: Total disruption of surgical wounds of the abdominal wall with reference to plasma proteinemia and plasma ascorbic acid, Am. J. Surg. 66:220, 1944.

2. Joergenson, E. J. and Smith, E. T.: Post-operative abdominal wound separation and evisceration. Am. J. Surg. 79:282, 1950.

3. Disruption of abdominal wounds: Collective review, Internat. Obst. Surg. 68:585, 1937.

4. D'Ingianni, V.: Wound disruption following cotton sutures, Am. J. Surg. 69:137, 1945.

5. Walton, F. E.: Prevention and treatment of wound dehiscence, Arch. Surg. 57:217, 1948.

6. Fallis, L. S.: The management of post-operative wound separation, Surg. 9:198, 1941.

7. Glenn, F., and Moore, S. W.: Disruption of abdominal wounds: Continuation of previous report, Surg. Gynec. & Obst. 72:1041, 1941.

8. Hinton, J. W.: Allergy as explained by dehiscence of wounds and incisional hernia, Arch. Surg. 33:197, 1938.

9. Singleton, A. O., and Blocker, T. G., Jr.: The problem of disruption of abdominal wounds and post-operative hernia. A review of 9,000 consecutive abdominal incisions, J.A.M.A. 112:122, 1939.

10. Totten, H. P.: Postoperative eventration, West. J. Surg. 46:305, 1938.

11. Mayo, C. W., and Maddison, J. L., Jr.: Separation of abdominal wounds, Arch. Surg. 62:883, 1951.

12. Bowen, A.: Post-operative wound disruption and evisceration: An analysis of 34 cases with a review of the literature, Am. J. Surg. 47:3, 1940.

13. Sustendal, G. F., and Collins, C. G.: Abdominal wound complications in obstetrics and gynecology, Obstet. and Gynec. 1:263, 1953.

DISCUSSION

Dr. Rodney G. Masterson, (Alexandria): Wound disruption is a disagreeable (and frequently a fatal) complication of abdominal incisions. Dr. Nix has given us an excellent analysis of a large series of cases. Since this is a continuation of the series of cases presented by Dr. Conrad Collins in his original study of 2,700 cases from the years 1946 to 1951 (which appeared in the Green Journal of Obstetrics and Gynecology), we are able to evaluate the continued careful handling of abdominal wound complications. Dr. Nix has re-emphasized the importance of early and deliberate exploration of wound complications as a surgical problem, thereby reducing mortality.

Wound disruptions are hazardous complications, and are usually a local manifestation of a generalized failure of the healing process. Local factors, with the exception of infections, are rarely of great significance. Urgent need for operative intervention precludes satisfactory preoperative preparation of the patient. Deaths following wound disruptions are usually due to factors other than the disrupted wound.

The utilization of various suture material is an interesting topic for discussion, but not a basis for major controversy. Standardization of a good technique, as utilized in this Tulane Teaching Service is the essential factor.

One important factor in the current ever-

lowering incidence of wound disruption appears to be the adoption of the transverse incision in both the lower and upper abdomen. I personally utilize the lower transverse incision whenever possible. But the longitudinal incision is indicated in many instances.

In such a large teaching service it is necessary to standardize a good technique for many operative procedures; therefore, there are enough disadvantages to the transverse incision to overshadow its advantages in such a large teaching service.

In the present day of antiseptic and aseptic surgery, clean surgical wounds are expected to heal without infection. But in any large series, the number of postoperative infections in clean wounds will always be in the percentage column of complications.

The degree to which wound infections contribute to disruption is debatable. In the first series of cases presented by Dr. Collins, there were 26 wound abscesses, or 30 per cent of wound complications were due to abscess. The preoperative abdominal preparation in these cases consisted of tincture of green soap, ether and merthiolate.

In the second series presented by Dr. Nix, the total number of abscesses was markedly reduced by 30 per cent. In this series the preoperative abdominal preparation was changed from tincture of green soap, ether and merthiolate, to phisohex and zepharin solution. Hexachlorophane (or G-11) used with either soap or a detergent has given us a new era in preoperative scrubs and skin preparation.

The efficiency of hexachlorophane has been established. A detergent with hexachlorophane is placed upon the skin for a period of ninety seconds. Then 9 brush strokes will accomplish the equivalent of approximately 150 brush strokes with the use of tincture of green soap and water. This has been a remarkable change in preoperative preparation.

There are many trade name soaps and detergents which contain hexachlorophane on the market, i.e., phisohex, septisol, germa-medica, dial soap, etc., and such a product should be used by the patient in her home before admission to the hospital both for vaginal and abdominal preps. We should take advantage of this prophylactic measure and instruct our patients accordingly in the use of these preparations, before admission to the hospital for surgery.

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SURGICAL CORRECTION OF CONGENITAL COARCTATION OF THE AORTA: A REPORT OF FIVE CASES *

CHARLES A. BESKIN, M. D. †
BATON ROUGE

In 1945, independent reports by Crafoord and Gross demonstrated that coarctation of the aorta could be treated surgically by resection and reestablishment of aortic continuity. Before that time, recognition of this anomaly was of academic interest only. Since then, hundreds of cases have been operated upon and the surgical treatment of coarctation of the aorta has received universal acceptance. The burden of this deformity is not usually manifest until early adulthood. However, it has been shown that 70 per cent of patients with this lesion die on or before their fortieth year. The most common cause of death is cerebral hemorrhage due to the markedly elevated cerebral hypertension. Since operative treatment removes the defect and establishes a normal anatomy, the rationale of the operation can be easily understood.

DIAGNOSIS

If the possibility of the presence of a coarctation is kept in mind, the diagnosis is not difficult. In childhood the patient may be completely asymptomatic. There is usually a systolic precordial murmur and hypertension in one or both arms. The finding of absent femoral pulsations or diminished blood pressure in the lower extremities generally confirms the diagnosis. As the patient grows older, one finds evidence of collateral circulation; there may be palpable intercostal or parascapular arterial pulsations or an audible bruit over the intercostal vessels. Notching of the ribs is often seen on a routine

* Presented at the Seventy-sixth Annual Meeting of the Louisiana State Medical Society, Alexandria, La., April 24, 1956.

† Active Staff, Our Lady of the Lake Hospital Active Staff, Baton Rouge General Hospital Consultant, Thoracic Surgery, Greenwell Springs Tuberculosis Hospital (Baton Rouge). Visiting Surgeon (Tulane), Charity Hospital (New Orleans).

chest film. In almost all cases, the coarctation is in the descending arch of the aorta just below the take-off of the left subclavian artery. It is usually just at or below the ligamentum arteriosum. In rare instances, coarctation may occur in lower segments of the aorta. The deformity is almost always of an hour-glass type, but in rare instances, may extend for several centimeters. There are usually premature atherosclerotic changes in the region of the obstruction. Aortography is not essential for diagnosis, but can be valuable in demonstrating the site of the obstruction and its extent when an atypical situation is suspected.

SURGICAL TREATMENT

The surgical treatment is simple in concept. Its purpose is the removal of an obstruction and the establishment of a normal channel of blood flow. Because resection and end-to-end anastomosis is the simplest and invites the fewest complications, it is understandably the ideal end to achieve. The latter can be carried out in almost all instances in the age group under eighteen. Because of this and the fact that the young aorta is pliable and elastic, the ideal age for surgery is early in the second decade. At this time, the aorta has reached near adult diameter and is easy to work with. However, if surgery is mandatory in the infant age group, it can be successfully carried out. Occasionally, coarctation of the aorta, either singly or in combination with other congenital defects, can cause severe embarrassment and failure in the infant group. Such a situation is demonstrated in one of the cases (A.W.M.) that are reported herein. In the adult age group, one usually finds an atherosclerotic, rigid aorta. It is often impossible to effect an end-to-end anastomosis and the use of a homograft is necessary. Because of the foregoing, a preserved homograft should be made available in all cases over the age of eighteen.

Complete dissection of the descending thoracic aorta, including its intercostal branches, is a requisite. This dissection is usually begun above the left subclavian artery and carried down through at least the four to five pairs of intercostal vessels below the coarcted segment. This makes it possible to elevate the aorta from its bed and provide adequate mobilization to effect a primary anastomosis. In general, the technique as practiced by Robert Gross has been found to be the most satisfactory by the author.

POSTOPERATIVE RESULTS

The postoperative results are generally highly satisfactory. In some instances, there is an unexplainable persistent elevated pressure in the presence of a known correction of the aortic block. However, in most cases the blood pressure returns to normal range and adequate circulation to the lower extremities becomes evident. In competent hands, the operation, though formidable, can be done with a negligible mortality risk.

The author has operated upon five cases* of congenital coarctation of the aorta in Baton Rouge, Louisiana since October 1953. Although not large, this series indicates that the anomaly is not the rarity it was formerly considered to be. The cases in the series range in age from three months to twenty-four years, and pretty well demonstrate the gamut of clinical findings and complications that occur in this condition. There was no mortality in the operated group, and all cases are presently well with significant and gratifying clinical improvement. Perhaps the most interesting of the five cases is that of an excision of a coarctation and anastomosis in a three month old infant under hypothermia. It demonstrates the observation that this condition is not an uncommon cause of cardiac failure in infants. There has been an increasing number of reports of late of operative repair in infants who would otherwise have died before they reached the optimal age for surgery. The higher mortality

* Two additional cases have been operated upon since this presentation—both males, ages 6 and 28. The latter required a graft to bridge the defect.

risk and the concern for the fate of an early anastomosis should not preclude an operation in this group when it becomes clear that relief of the aortic block is of immediate and vital importance. The other four cases will also be discussed in some detail, since they demonstrate the variety of features that characterize this condition in the different age groups.

CASE REPORTS

Case No. 1.—Baby A.W.M., a white male infant, had been noted to have respiratory difficulty and tachycardia since birth. When he was six weeks of age, the family noted an inspiratory retraction of the lower costal margins, exertional pallor and marked fatigability during feeding.

X-ray studies disclosed the heart to be enlarged, with prominence of the left ventricle and left auricle. The lung fields were engorged.

EKG revealed a sinus tachycardia of 170, and evidence of left ventricular strain. The femoral pulses could not be felt, and the blood pressure in both arms was recorded as 190/110. The radial pulses were bounding.

The liver was slightly enlarged. Routine blood studies and urinalyses were normal.

It was felt that the child probably had a coarctation with a possible associated patent ductus. At age three months, the child was observed for two weeks and digitalized. He improved only minimally on full digitalization and had to be kept in an oxygen tent throughout this time. On November 20, 1955, surgery was undertaken under endotracheal anesthesia and induced hypothermia. The child was lowered into an ice bath, and the rectal temperature reduced to 84 degrees Fahrenheit over a period of 45 minutes. The infant was then placed in right lateral decubitus position, and no further anesthetic mixture was given.

An obvious vascular abnormality was found in the left gutter. A classical coarctation deformity was found about 8 mm. below the take-off of an enlarged left subclavian artery. The intercostal vessels entering the lower aortic segment were not enlarged. Just 2 mm. above the coarctation was a widely patent ductus with a lumen of at least 3 to 4 mm. It was obvious that almost all of the blood flowing through the descending arch was being shunted through the ductus to the pulmonary circuit. The ductus was divided and the coarcted segment removed. A primary anastomosis was made without difficulty, using interrupted everting sutures of 5-0 silk. Following anastomosis, there was a bounding flow through the realigned aorta.

During the operative procedure, which lasted

Coarctation of the Aorta -

PATIENT	BRACHIAL PRESSURE PREOP.	BRACHIAL PRESSURE POSTOP.	OPERATION	RESULT
S.A wf 18yrs.	190/110	115/80	Resection Prim. Anast. 8-25-54	Good
P.M.T. wm 24yrs.	190/115	140/90	Resection Prim. Anast. 10-22-54	Good
J.R.L. wm 24 yrs.	210/110	130/90	Resection Homograft (5cm.) 2-6-55	Good
J.F.S. wm 11yrs	170/100	120/80	Resection Prim. Anast. 6-8-55	Good
A.W.M. wm 3mos	190/120	140/90	Resection Prim. Anast. Hypothermia 11-20-55	Good

Figure 1

two and a half hours, the rectal temperature dropped to 76 degrees Fahrenheit. Immediately following wound closure, the child was rewarmed with a warm water bath and hot water bottles. The child's temperature returned to normal over a period of about six hours. Recovery from then on was uneventful except for an atelectasis of the left lung the morning after surgery, which responded to laryngoscopy and suction. The child's progress since surgery has been most gratifying. He has gained six pounds in weight to date and appears to be normal in every respect. There are easily palpable femoral pulsations, with a recorded femoral pressure of 140/90. The brachial pressures have dropped to a level between 130 and 140 systolic. Though hypothermia is not essential in operating upon these infants, it is the opinion of the author that this particular case was greatly benefited by the decreased oxygen demand during surgery.

Case No. 2.—J.F.S., a white male of 11 years, had been noted to have absent femoral pulsations and mild brachial hypertension since the age of five. He had been observed since then by his father, a physician, and was noted to have progressive increase in his brachial pressure to the level of 178/100 shortly prior to surgery. The boy's only other symptom was weakness of both legs after running. There were no headaches and no evident cardiac enlargement by either x-ray or EKG. On examination, the child presented a rough, coarse systolic murmur at the apex and widely transmitted.

At surgery, the patient was found to have a classical coarctation about 1 centimeter below the take-off of the left subclavian artery. In freeing up and dividing the ligamentum arteriosum, it was surprisingly found to have a lumen of about 2 mm. A primary resection was performed and an end-to-end anastomosis using interrupted everting 5-0 silk was made. The aorta was easily mobilized, and the child withstood the procedure without incident. He left the hospital on the ninth postoperative day, and four months after surgery, has a brachial pressure of 122/80, with easily palpable femoral pulsations and a femoral pressure of 140/90. His ischemic leg pains have disappeared, and he has been noted to have an increase in the girth of his legs.

This case demonstrates the ease and relative safety that can be offered a patient at this ideal age. At this age, the aorta is always pliable, elastic, and of sufficient diameter to provide adequate flow during later years.

Case No. 3.—S.A., a white married female of 18 years, had a known heart murmur since birth. For the previous five years she had complained of headache, leg weakness, and had been noted to have a mild hypertension of 150/90. A definitive diagnosis was not made until 1954, when, following a three month miscarriage, her obstetrician noted that she had absent femoral pulsations. At that time, her brachial pressure at rest was recorded as 170/100. An x-ray of her chest showed slight left ventricular hypertrophy and definite rib notching. Surgery was advised and carried out at the Baton Rouge General Hospital on August 25, 1954. The patient was found to have a characteristic hour-glass deformity about one centimeter below the obliter-

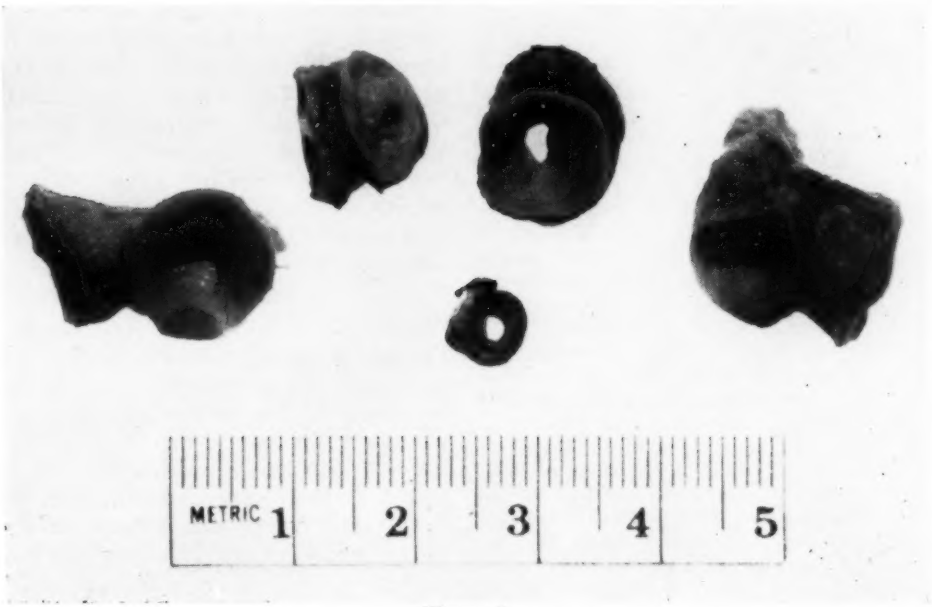

Figure 2

ated ligamentum arteriosum. There was marked enlargement of the intercostal vessels entering the aorta below the coarctation. The coarcted segment was resected, and an end-to-end aortic anastomosis performed using an interrupted everting 5-0 silk technique. The patient was discharged from the hospital on the tenth postoperative day. Her brachial pressures now range between 130 and 138 over 90. She has good femoral pulsations. She became pregnant again one year ago, and three months ago was delivered of a normal full-term pregnancy. She had a completely uneventful course through her pregnancy and her pressure never ran above 170 systolic during the last trimester.

This case has been particularly gratifying, since pregnancy is not well-borne by the uncorrected coarctation, and if not spontaneously aborted often leads to aortic rupture or cerebral hemorrhage.

Case No. 4.—P.M.T., a married white male of 24 years, had a known heart murmur since age ten. He was taken into the service but two months after induction, a diagnosis of coarctation of the aorta was made and surgery advised. He refused surgery and was given a medical discharge. Since then he had had progressive complaints of cold feet, headaches, and moderate dyspnea on exertion. Physical examination revealed a bilateral brachial pressure ranging up to 210/110 after mild exercise. Femoral pulsations were absent. A routine chest x-ray showed characteristic rib notching. There was a soft systolic murmur best defined at the base of the heart. At surgery on October 15, 1954, the patient was found to have a rather long coarcted segment, with an abnormally narrow proximal aortic arch. The entire aorta was freed and an end-to-end anastomosis made without using the available homograft. The first pair of intercostal arteries below the coarctation were aneurysmal in size. These were ligated and divided. The aorta was clamped for a period of forty minutes during the anastomosis. Following surgery, the patient was found to have flexor weakness of his left leg. It was assumed that this was due to interference with the collaterals. The patient made an uneventful recovery but required the use of a walking stick because of his left leg weakness for a period of about three months. He is now able to get about without a limp and his brachial pressures range between 130 and 140 over 95. His femoral pressures are low (90/50) but a faint femoral pulsation can be felt.

Case No. 5.—J.R.L., a married white male of 24 years, had been told that his blood pressure was high since the age of eight and that he had "some sort of heart disease". A heart murmur had also been heard since that time. For the previous four years he had complained of dyspnea on exertion and headache. A diagnosis of coarctation of the aorta was made by his family physician during a routine physical examination for life insurance. At that time his brachial pressure was recorded as 178/110. He had definite pulsations of his intercostal arteries, and a systolic murmur in the second left interspace. X-rays revealed left ventricular enlargement and classical rib notching. His femoral pulsations were absent and no blood pressure was recorded in his femoral artery.

Surgery was advised and carried out on February 7, 1955. He was found to have a rather long coarcted segment in the classical location one centimeter below the take-off of the left subclavian artery. After resecting the coarcted segment, despite adequate mobilization of the distal portion, it was impossible to bring the ends of the aorta together. A preserved homograft, made available prior to surgery, was inserted to bridge the defect. The homograft measured 5 cm. in length and an interrupted everting 5-0 silk technique was used. The patient made a very smooth recovery and was discharged from the hospital on his tenth postoperative day. He is now fourteen months after surgery and his brachial pressures range from 130 to 138 over 90. He has noted a complete disappearance of all his previous symptoms, has full palpable femoral pulsations and a femoral blood pressure of 160/100.

SUMMARY

The surgical correction of congenital coarctation of the aorta is a procedure now widely accepted and well standardized. Resection of the coarcted segment and end-to-end anastomosis is the ideal management; in some cases, particularly in the older age group, a homograft is necessary to bridge the defect. The diagnostic criteria are fairly simple if the possibility of the presence of the anomaly is kept in mind. In most instances, surgery can offer complete correction of the defect. In competent hands, the operation can be performed with a negligible mortality risk.

Five consecutive cases of surgical correction of coarctation of the aorta performed in Baton Rouge during the past two years are presented. There has been no mortality in the operated group, and all cases are presently well with significant clinical improvement. The cases range in age from three months to twenty-four years, and illustrate the particular problems presented in various age groups.

A preserved homograft was used in

one of the five cases, and the advisability of having a prepared graft available in the over eighteen age group is stressed.

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THE CLINICAL RELATIONSHIP OF THE ADRENAL CORTEX TO DIABETES MELLITUS *

DOUGLAS GORDON, M. D. †
BATON ROUGE

In a discussion such as this, it seems advisable to define diabetes mellitus. An excellent definition is the one by Jackson:[1]

"It is an innate, obscure disorder of carbohydrate metabolism, which manifests itself in curious, diverse ways — in the hypertrophied islets of overweight stillborn babies; in microaneurysms in the retina; in cardiac infarction in the female; and in excessive tallness in the child and obesity in the mother—apart from the clinically obvious glycosuria, ketosis, and hyperglycemia."

Jackson believed that diabetes may be hereditary, congenital, or acquired, and may be present for many years before becoming clinically obvious, in fact, from birth in many instances. If diabetes is considered to be this type of disease, many factors will enter into production of symptoms, as well as treatment of the patient. One of the most interesting factors which has an influence on carbohydrate metabolism and upon diabetes mellitus is adrenal cortical function. Many methods have been used in attempting to assess the relationship between the adrenals and diabetes. Studies which have been performed include adrenalectomy and hypophysectomy in normal and pancreatectomized animals and administration of anterior pituitary extract-growth hormone, ACTH and cortisone.

EXPERIMENTAL OBSERVATIONS— ADRENALECTOMY

Adrenalectomy was first shown by Long and Lukens[2] to alleviate the diabetes that developed in laboratory animals following pancreatectomy. They[3, 4] later confirmed and enlarged upon this observation and drew the following conclusions. Adrenalectomy is followed by a decrease in concentration of carbohydrate in the blood and liver, but the extent of decrease in muscle glycogen is questionable. The respiratory quotient usually remains normal. Following fasting, hypoglycemia and extreme depletion of liver glycogen develops. Their animals were hypersensitive to small doses of insulin, and in addition, there was a decrease in the urinary excretion of glucose, nitrogen, and ketones. Following adrenalectomy, the result of the intravenous glucose tolerance test remained normal or showed increased tolerance. The peripheral oxidation of carbohydrate was unchanged, as measured by radioglucose technics.[5] Since the intensity of the diabetes could be restored by adding adrenal cortical hormones, which also resulted in replacement of liver glycogen stores, it was believed that adrenalectomy probably resulted in defective conversion of protein to carbohydrate or gluconeogenesis. Ingle[6] thought that in many of the early experiments, the alterations in carbohydrate, fat and protein metabolism could possibly have been due to inadequate feeding of the animals, shock, or infection. He showed that in adequately supported animals, forced fed and maintained on a 1 per cent solution of sodium chloride, adrenalectomy alone may result in no change in the blood, tissue or liver carbohydrate, nitrogen balance or fat metabolism. If these animals were forced fed on a medium carbohydrate diet, no difference occurred in the nitrogen excretion of the adrenalectomized as compared with the sham-operated control. Furthermore, in a partially pancreatectomized rat given a constant food intake, mild glycosuria could be abolished by adrenalectomy without decreasing the amount of urinary nitrogen excretion.[7] These and other experiments showed that it is possible for an adrenalectomized rat to metabolize large amounts of fed protein when kept in

* Presented at the Seventy-sixth Annual Meeting of the Louisiana State Medical Socitey, Alexandria, La., April 25, 1956.

† Department of Medicine, Tulane University School of Medicine and the Endocrine Research Laboratory of the Alton Ochsner Medical Foundation, New Orleans.

good condition with sodium chloride intake.

Pincus and Elmadjian [8] summarized the current concept regarding these aspects of adrenalectomy by stating that decreased hepatic gluconeogenesis had been definitely shown to occur in adrenalectomy, although there is still disagreement as to the influence on glucose utilization by peripheral tissues.

HYPOPHYSECTOMY

With regard to the influence of the pituitary upon carbohydrate metabolism, Houssay [9] demonstrated very clearly that hypophysectomy results in pronounced amelioration of pancreatic diabetes. Prior to these experiments, Olmsted and Logan [10] had shown that insulin convulsions could be produced in decerebrated cats whose pituitaries had been removed, but not in animals with intact pituitaries given the same dose of insulin. Houssay further showed that removal of the pancreas in hypophysectomized dogs or toads resulted in less urinary excretion of glucose, nitrogen and ketones than that seen after pancreatectomy alone. Therefore, if the pituitary were in place in these pancreatectomized animals, the conversion of protein to carbohydrate increased. Lukens [4] pointed out that in the pancreatectomized cat, abscesses rapidly developed in the incision, whereas, if the pituitary, as well as the pancreas, was removed, such abscesses healed completely. Apparently, although there is an increase in blood sugar and insulin is absent in both groups of animals, removal of the pituitary results in a decreased breakdown of protein and fat. Lukens [4] further showed that pancreatectomized cats will lose 300 to 400 Gm. in four days of survival; whereas if the pituitary is removed as well as the pancreas, the same amount of weight will be lost over a period of two months. Therefore, the lack of insulin causes loss of weight, but this is definitely accelerated if the adrenal and pituitary are functioning and this is related to the adrenal catabolic influence.

ANTERIOR PITUITARY AND GROWTH HORMONE ADMINISTRATION

With respect to administration of anterior pituitary extracts, Houssay [9] showed, in 1932, that this material injected into normal animals could result in temporary hyperglycemia and glycosuria. Young [11] produced permanent diabetes by giving normal dogs anterior pituitary extract. In addition, this substance worsened the diabetes of Houssay's animals in which both pituitary and pancreas had been removed. In cats and dogs, partial pancreatectomy, administration of pituitary extract, growth hormone, or massive doses of glucose produced similar changes in the beta cells of the pancreas resulting in atrophy.[4] If the hyperglycemia of these animals were prevented by administration of phlorhizin or insulin concomitantly, the permanent microscopic changes as well as permanent diabetes did not develop.[12, 13] Therefore, the microscopic changes were secondary to the hyperglycemia and not directly the result of administration of the growth hormone or pituitary extract. Young [14] has since related the diabetogenic activity of anterior pituitary extract to the more purified action of growth hormone.

Administration of growth hormone promotes many changes.[4] Among these are positive nitrogen balance, growth, depression of glucose uptake by isolated diaphragm muscle, and prevention of further loss of muscle glycogen in a fasted hypophysectomized animal. It makes alloxan diabetes worse, and can produce permanent diabetes in the dog or cat, although it is ineffective in intact rats. Engel and associates [15] showed that growth hormone became diabetogenic in the intact force-fed rat if ACTH were also given. The work of deBodo and Sinkoff [16, 17] has further elucidated growth hormone function. They observed that if the pituitary were removed, insulin sensitivity was greater than if only the adrenal cortices were removed; this shows that there is a pituitary factor, separate from ACTH, that is concerned with insulin sensitivity. Administration of thyroxin or prolactin resulted in only partial abolition of the insulin hypersensitivity of

the hypophysectomized dog; whereas administration of growth hormone diminished and, with continued treatment, completely abolished this hypersensitivity. Therefore, the anti-insulin action of growth hormone could occur in the adrenalectomized, hypophysectomized dog and therefore could exert its diabetogenic action without going through the adrenal. There is still some question as to whether or not growth hormone actually interferes with peripheral utilization of carbohydrate.

EFFECTS OF ACTH AND CORTISONE

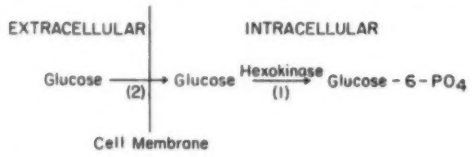
Administration of ACTH and cortisone in experimental animals has led to further elucidation of the role of the adrenal in carbohydrate metabolism. Long and Lukens [2] alleviated experimental pancreatic diabetes by removing the adrenal cortex. Administration of the adrenal cortical extract conversely resulted in intensification of the diabetes. Therefore, a definite relationship between diabetogenic and adrenal activity was demonstrated. Venning, Kazmin and Bell [18] devised an assay for adrenal cortical hormones based on the fact that livers deplete of glycogen developed in fasted mice given only salt and water. When these animals were given cortisone, an increased content of liver glycogen would develop which was directly proportional to the amount of cortisone administered. Further studies revealed that administration of adrenocortical steroids could produce "steroid" diabetes in the normal force-fed rat.[3, 6] This diabetes was typically mild although the animals were hyposensitive to insulin. Urinary excretion of nonprotein nitrogen increased in association with increased liver glycogen, which was good evidence in favor of gluconeogenesis. When food was withheld, glycosuria disappeared although the negative nitrogen balance continued. The diabetes was characteristic in that the glycosuria that developed as a result of steroid treatment would gradually decrease over a period of time and could be reestablished if a further increased dose of steroid were given. The glycosuria disappeared when the steroid injections were discontinued. The increase in glycosuria following treatment was greater than could be accounted for by increase in urinary nitrogen excretion and therefore Ingle [6] thought that the adrenal cortical hormones either affected conversion of fat to carbohydrate or inhibited carbohydrate utilization. Segaloff and Many [19] showed that ACTH in extremely large doses given to intact rats greatly increased excretion of glucose as well as ketosis but did not affect urinary nitrogen. This is further evidence that adrenal cortical function increases conversion of fat to carbohydrate.

The newer adrenal cortical steroids vary in their activity upon carbohydrate metabolism as compared with their salt and water effects.[20] Aldosterone and the synthetic fluorinated compounds have much greater salt retaining properties than carbohydrate effects whereas the Delta [1] series, such as prednisone, shows greater carbohydrate effects than sodium retaining activity. The 2-methyl analogues seem to be more potent than the nonmethylated steroids with respect to both the glycogen depositing action and the sodium retaining property.[21]

In addition to the metabolic changes resulting from administration of ACTH and cortisone in experimental animals, one of the most interesting developments in the last few years has been the experimental production of capillary disease of the retina and the renal glomerulus. In 1952, Becker [22] reported the appearance of retinal micro-aneurysms as well as renal capillary changes, suggestive of the Kimmelstiel-Wilson type, in alloxan diabetic rabbits treated with ACTH. Alloxan alone did not produce these lesions. Bloodworth and Hamwi [23] confirmed these results by demonstrating both nodular and diffuse glomerular lesions in normal as well as alloxanized rabbits treated with cortisone. The degenerative vascular lesions seen in diabetes have been thoroughly reviewed recently by Ricketts [24] with

a discussion of the possible relationships involved.

The manner in which the adrenal hormones influence insulin activity is not known. Levine and Goldstein [25] offered a



(After Levine and Goldstein)

Figure 1.—Possible sites of hormone action in glucose utilization.[25]

hypothesis which they believed explains the major metabolic consequences of insulin lack or excess. As shown in Figure 1, there are two possibilities, either of which would explain most of the experimental evidence pointing to the insulin effect. One is inhibition of the hexokinase reaction, and the other, the possibility of activating a mechanism whereby glucose entered the cell. Most of the prior experiments would show the same effect, whether acting upon the hexokinase activity or the cell permeability; in one instance it would be a pulling effect and in the other a pushing effect on glucose. Therefore, a nonutilizable carbohydrate was used as a means of distinguishing these functions. Cortisone was shown to have produced almost complete insulin resistance with regard to glucose, but not galactose, which is a nonutilizable carbohydrate. They believed that activation of a mechanism of entry into the cell is the primary effect of insulin, and that once in the cell, the pathway of metabolic function continues whether insulin is present or not. They concluded that pituitary and adrenal cortical secretions have some inhibitory influence on glucose uptake by muscle and they then supposed that the pituitary and possibly adrenal cortical hormones inhibit the hexokinase activity but do not operate upon step 2, which would be the permeability of the cell to the substance.

It should be mentioned that the adrenal may be playing a permissive role in diabetes. In a recent editorial, Engel,[26] who pointed out the evidence in favor of this, mentioned that subthreshold types of stress alone may produce no response or a delayed response; whereas a subthreshold stress, plus small amounts of adrenal cortical extract, will produce a protein catabolic reaction. Therefore, the adrenal cortical hormone is necessary for the organism to respond, but is not primarily responsible for this type of stress reaction. Furthermore, hypersecretion of adrenal steroids does not seem to be necessary to initiate the metabolic response, but a high level of secretion may be necessary to sustain it. This would explain the high levels of adrenal cortical hormones secreted during periods of stress.

CLINICAL OBSERVATIONS

The next question that comes to mind is: what role do the pituitary and adrenal play in human diabetes, either in causing it or possibily in aggravating it during periods of infection, stress and emotional upset? Secondly, is experimental diabetes, such as the type just discussed, truly the diabetes seen clinically or is this just a defect in carbohydrate metabolism without the other associated manifestations of diabetes? Since most cases of clinical diabetes are due to an actual insulin deficiency, it is well to consider whether the adrenal plays a necessary role in maintaining this metabolic disturbance, whether an adrenal disturbance is actually present and if so, just how often subclinical adrenal disturbances will lead to the carbohydrate defect seen in diabetes, without the stigma usually seen in clinically evident adrenal disease. Although there is no definite answer to these questions at present, it is of interest to consider the experimental work on patients with respect to the adrenal's role in carbohydrate metabolism.

ACTH AND CORTISONE ADMINISTRATION

The effect of ACTH and cortisone in the human has been thoroughly investigated within the last ten years and considerable information has accumulated. Large doses of ACTH given to normal people will induce a state of diabetes, in

other words, hyperglycemia as well as glycosuria, which is relatively resistant to insulin.[27] In some normal people, glycosuria develops without hyperglycemia; this indicates decreased renal reabsorption of glucose. Conn and associates [27] showed that there is a decrease in the level of glutathione in the blood associated with induction of steroid diabetes in man. Since they noted an increase in uric acid excretion as well, which has been thought to be related to alloxan-like compounds, they suggested that possibly the decrease in glutathione is in some way a decrease in the protective mechanism of the pancreas against any alloxan-like effect. The average dose of cortisone that most patients receive is usually extremely small and, apparently for that reason, only slight or no changes at all in carbohydrate metabolism occur. Rarely is more than a trace of sugar found in the urine even after meals. The glucose tolerance test is more sensitive than the fasting blood sugar and was found to be abnormal in 4 of 12 patients treated with cortisone. However, 2 of these 4 had a family history of diabetes. There have been some instances in which after prolonged treatment with cortisone there was still no evidence of impairment of glucose tolerance. Sprague and coworkers [28] believed that this might indicate that in some persons cortisone had made manifest a latent diabetes. Burns, Engel and others [29] showed that a single dose of cortisone (200 to 500 mg.) given to a normal person would result in significant elevation of the blood sugar four hours later as well as impairment in the glucose tolerance. The glucose insulin or insulin tolerance alone was not impaired by the same treatment and indicated no evidence of decreased sensitivity to insulin. Intravenous administration of compound F produced no hyperglycemic effects in a four hour test, but did show a decrease in the standard glucose tolerance.[30] As a result of serum phosphorus changes, indicating normal glucose utilization, it appeared that insulin sensitivity remained normal, that there probably was an increase in protein conversion to carbohydrate, and possibly an increase in glucose utilization. There seems little doubt that cortisone increases conversion of protein to carbohydrate as evidenced by the increased urinary nitrogen and increased glycogen stores seen in patients treated with cortisone. However, studies performed on diabetic persons while on a carbohydrate free diet showed that cortisone increased the glycosuria in excess of that explainable by gluconeogenesis from protein alone; therefore, fat must be converted to carbohydrate in order to explain the full diabetogenic effect of adrenal steroids.[31]

Becker [22] reported appearance of retinal capillary aneurysms similar to those seen in persons with diabetes in 2 patients given ACTH intravenously for sarcoidosis. These changes disappeared when ACTH was discontinued. This last observation of Becker has brought further attention to the possibility of the adrenal's role in producing capillary disease in diabetes mellitus.

Administration of ACTH to diabetic persons results in an increased degree of ketosis and hyperglycemia, decreased utilization of carbohydrate and increased excretion of carbohydrate in the urine.[31, 32] The ratio of dextrose to nitrogen in the urine suggests that fat as well as protein is being converted to carbohydrate. Diabetic patients require more insulin during treatment than they ordinarily do. A more interesting observation by Kinsell and coworkers [31] is that the insulin resistance during ACTH and cortisone treatment could be prevented by administration of potassium salts. Conversely, insulin resistance occurred only in patients who exhibited some interference with sodium or potassium stores in the body. It is interesting that in no diabetic patient treated with either ACTH or cortisone did the increased severity of the diabetes continue after discontinuance of use of the hormone. In two instances, less insulin was required than before treat-

ment and it is possible in these that ACTH or cortisone could have benefited insulin resistance secondary to some allergy. Conn and Fajans [33] stated that development of decreased carbohydrate tolerance during ACTH treatment depends upon dosage and duration of administration of ACTH, whether the index is the fasting blood sugar or the more sensitive glucose tolerance test, and the patient himself. In addition, there might be interference with the ability of the pancreas to produce the necessary increase in insulin supply.

Fajans and Conn [34] did glucose tolerance tests on 252 healthy relatives of diabetic patients and on 50 normal control subjects, both before and after oral administration of 100 or 125 mg. of cortisone. Twenty-four percent of the nondiabetic relatives showed pronounced reduction in carbohydrate tolerance during the cortisone glucose tolerance test as compared with only one of the 37 controls. This type of test would be of value in detecting the prediabetic patient although at present there is certainly no means of preventing progression of the condition to true clinical diabetes, except possibly by maintaining normal weight.

McQuarrie [35] called attention to use of ACTH in idiopathic spontaneous hypoglycemia of infants and children. It was thought at first that the deficiency of alpha cell secretion of glucagon would possibly account for the hypoglycemia in these children, but other factors are apparently important. These children have a hypoglycemia suggestive of excessive insulin production in the fasting as well as in the postprandial state. As McQuarrie pointed out, these children do not have pancreatic islet tumors but definite cerebral damage will develop in them if the condition continues over a period of years. Since the condition subsides in many as they grow older, everything should be done to prevent damage to the brain from the hypoglycemia. For that reason ACTH has been used successfully and in several instances it has been discontinued after several months. Conn and Seltzer [36] in a discussion of organic hyperinsulinism or hypoglycemia, mentioned that ACTH can be used in the preoperative preparation of these patients. This elevates the blood sugar and also prevents the peculiar type of hyperthermia which has been noted following surgical removal of pancreatic adenomas. If the patient refuses to submit to operation, or if there is too great a risk for operation, then hydrocortisone in large doses can be used and will maintain the fasting blood sugar levels.

CUSHING'S SYNDROME

One would expect in Cushing's syndrome due to increased adrenal cortical secretion a definite interference in carbohydrate metabolism. Knowlton [37] found this to be true in 94 per cent of the patients having a carbohydrate tolerance test. However, of the 38 patients, only 24 per cent showed overt diabetes. The average fasting blood sugar was normal. He believed that severe carbohydrate disturbances in Cushing's syndrome are rare as evidenced by only one instance of acidosis in a review of 189 cases in the literature. Cope and Raker [38] reported similar observations. Patients having an insulin tolerance test revealed insensitivity to insulin. Sprague and coworker's [28] patients with Cushing's syndrome showed, in addition to the other findings, a negative nitrogen balance even when glycosuria was minimal or absent, and during fasting the diabetes became mild.

Balfour and Sprague [39] reported a case of Cushing's syndrome in which, despite 145 units of insulin a day, excessive glycosuria continued, but there was no evidence of ketosis. Following removal of the adrenocortical tumor, there was no further evidence of diabetes over a period of eight years. Many similar instances have been reported. Thus, this type of steroid diabetes is usually reversible if the lesion can be removed. It is similar to that seen in patients given excessive amounts of ACTH or cortisone in that the fasting blood sugar level is frequently

normal while the glucose tolerance test shows abnormalities, insulin resistance is present, a negative nitrogen balance will occur despite absence of glycosuria, and lastly, the glycosuria tends to decrease in the presence of fasting.

ACROMEGALY

The carbohydrate studies done in patients with acromegaly have shown a similar type of diabetes in that it is usually mild, insensitive to insulin and, if the pituitary lesion responsible for the acromegaly becomes inactive either spontaneously or with treatment, the diabetes becomes milder or even disappears.[39] Balfour and Sprague [39] reported a case of an acromegalic patient with a blood sugar of 384 mg. per cent, requiring 62 units of insulin daily who, following roentgentherapy to the pituitary tumor eventually needed no further insulin. Davidoff and Cushing [40] in 1927 reported only 12 per cent of their 100 acromegalic patients had diabetes. In another series [41] only 26 per cent had diabetes, which was mild, disclosed only by glucose tolerance tests and occurred more frequently in relatives of diabetics. Most cases are controlled by diet and in some instances small doses of insulin, although occasionally the severity of the diabetes changes rapidly.[42] Diabetic coma, gangrene, and other vascular complications rarely occur. Therefore, the mild diabetes seen in acromegaly is rarely a clinical problem.

PITUITARY INSUFFICIENCY

The development of pituitary failure or Simmond's disease in diabetes has received considerable attention in the literaature.[43, 44] Martin and Pond [43] found 11 cases in which the diabetes mellitus was alleviated by the onset of pituitary insufficiency. This has been referred to as the Houssay phenomenon in man. Most of the patients had moderately severe diabetes, requiring insulin therapy, who, following development of pituitary failure, began to notice frequent insulin reactions and resultant decrease in insulin requirement. Several discontinued insulin altogether. Harvey and de Klerk [44] mentioned two diabetic persons in whom pituitary failure developed so that insulin therapy was no longer required, but they continued to show a diabetic glucose tolerance curve. The most interesting case of this kind was reported by Poulson.[45] This was a woman, aged 30 years, who had had diabetes for twenty years and had severe diabetic retinopathy. Following postpartal pituitary failure, the insulin sensitivity increased and the patient no longer had a tendency to ketosis. Approximately four years later the retinopathy had decreased considerably and six years after development of pituitary failure, diabetic retinopathy was no longer present. Since the work of Becker [22] showed the influence of ACTH and cortisone upon the development of diabetic retinopathy, the reaction in this patient suggests a possible therapeutic approach —hypophysectomy.

Luft, Olivecrom and Sjogrn [46] have probably had the greatest experience in the use of hypophysectomy in diabetes. They reported 7 cases, all of which had chronic juvenile diabetes, with progressive malignant vascular complications. Only patients who were expected to die or become blind were selected for treatment. The insulin requirement preoperatively varied from 24 to 60 units daily and postoperatively it was 0 to 8 units daily. The effect on the vascular complications was difficult to assess at the time of their report, since all patients had far advanced disease at the time of operation. In one there was no further progression for one year. The hypertensive patients became normotensive and, in 2 patients, chronic albuminuria disappeared. Kinsell and coworker's [47] experience with hypophysectomy is similar. They pointed out that the insulin hypersensitivity that developed could be balanced by the judicious use of cortisone. This procedure is still in the experimental stage, and further work will have to be done before its therapeutic role can be properly evaluated.

ADRENAL DEFICIENCY

It was noted by Thorn and associates,[48]

in 1940, that patients in whom Addison's disease developed showed the similar changes expected from animal studies. Depletion of liver glycogen, decreased urinary nitrogen excretion, increased sensitivity to insulin and development of hypoglycemia during episodes of fasting all occurred. These changes can be corrected by administration of cortisone.[28] In the adrenalectomized patient hypoglycemic reactions do not occur and the glucose tolerance remains normal as long as the patient receives adequate hormonal maintenance.[49] Two of the patients adrenalectomized by Dao and Bergenstal [49] had diabetes. They required the same amount of insulin postoperatively as they did preoperatively if maintenance cortisone therapy was continued. There have been many instances of Addison's disease developing in preexisting diabetics. As would be expected, the insulin sensitivity greatly increased. Many patients who had been taking 30 to 40 units of insulin daily began to have increased insulin reactions to the point where even 3 or 4 units of insulin could produce a hypoglycemia.[39] This was corrected with either adrenal cortical extract or cortisone. Conn and Fajans [33] pointed out that patients with Addison's disease manifest a definite inability to fast without hypoglycemia, a greatly increased insulin sensitivity, and symptoms of hypoglycemia at higher levels of blood sugar than when corticoids are present. Cortisone in physiologic amounts reverses these effects. Cortisone replacement therapy in patients with Addison's disease and diabetes intensifies the diabetic state but the patient has fewer symptoms of adrenal cortical insufficiency and the diabetes is easier to control although more insulin is required.[33] Green and associates [50] first induced adrenal cortical insufficiency as a therapeutic measure in diabetes when they performed bilateral adrenalectomy in a patient with malignant hypertension and diabetes. The insulin requirement fell to one-fourth of the preoperative dosage and hypoglycemia occurred if an overdose of even 3 to 5 units of insulin was given. Adrenalectomy was also performed by Wortham and Headstream [51] in 7 patients with advanced vascular disease. Two patients showed mild reversal of retinal changes, return to normal blood pressure, and decrease in protein excretion. In 3 patients the preoperative vascular manifestations progressed no further. In general, the favorable results have been in inverse proportion to the severity of the vascular disease preoperatively. Adrenalectomy should be considered in the same experimental stage as hypophysectomy and should be limited to patients with vascular disease and rapidly progressive renal deterioration. The value of this procedure has yet to be proved.

ADRENAL FUNCTION IN DIABETICS

Studies of adrenal cortical function in diabetic patients in general reveal no increased adrenal function as evidenced by normal 17-ketosteroid and glycogenic corticoid values.[52] Patients during diabetic acidosis were found to have elevated urinary excretion of corticosteroids which was somewhat related to the severity of the diabetic acidosis.[53] However, in a patient followed after intentional withdrawal of insulin therapy the urinary corticoids did not become demonstrably increased until mild acidosis was clinically apparent. Therefore, this adrenal activity was a late phenomenon, secondary to and not responsible for the diabetic acidosis. On the basis of eosinophile responses it has been suggested that adrenal cortical unresponsiveness exists in diabetic persons, possibly secondary to the chronic stress of the diabetic condition.[54] Freeman and associates [55] found the free-reducing and hydrocortisone-like steroids in human plasma to be within normal limits in 8 diabetic persons. Walloch and coworkers [56] reported elevation of 17-hydroxy-corticosteroid levels in plasma in a patient with diabetic acidosis and return of these levels to normal with response to therapy. However, in patients who died, the blood level remained elevated until death. This suggests that increased adrenal

cortical activity is present in diabetic acidosis, but is the result rather than the cause. In addition, they had one patient with relative adrenal insufficiency in whom acidosis developed without an increased blood corticoid level. This further suggests that adrenal cortical increased activity is secondary. Izzo and Eilers [57] measured the total reducing corticosteroids and 17-ketosteroid excretion in patients with diabetes before and after cortisone administration. Significant increased excretion occurred in diabetic persons but not in normal controls; this suggests the possibility of altered steroid metabolism in diabetes mellitus.

TABLE 1
CLINICAL APPLICATION OF ADRENAL INFLUENCE ON CARBOHYDRATE METABOLISM

A. Diagnostic	
	Possibility of Adrenal Diabetes
	Possibility of Pituitary or Adrenal Failure Pre-diabetic
	Diabetes Mellitus or Cushing's Syndrome
B. Therapeutic	
	Side Effects of Cortisone Treatment
	Spontaneous Hypoglycemia
	Adrenalectomy or Hypophysectomy in Diabetes

CLINICAL IMPLICATIONS

Table 1 summarizes some of the possible diagnostic and therapeutic applications of the adrenal-carbohydrate relationship. Steroid diabetes should be considered for diagnostic purposes in all diabetic patients, particularly if the insulin requirement is high. A diabetic patient whose insulin requirement suddenly decreases and in whom hypoglycemic reactions begin to occur for no apparent reason should be considered to have possible pituitary or adrenal failure. The prediabetic patient is of interest at the present time and can be recognized if the cortisone-glucose tolerance test is employed, although no therapeutic measures are yet available for this peculiar syndrome. The differential diagnosis between diabetes mellitus and Cushing's syndrome is frequently a problem, particularly in an obese, hypertensive, diabetic, slightly hirsute individual. Many patients with Cushing's syndrome will have the other associated finding of osteoporosis, vertebral fractures, purple striae, amenorrhea and muscular atrophy. However, in atypical instances, the urinary excretion of corticosteroids will be the deciding factor. Since there is no good experimental evidence that indicates increased adrenal function in uncomplicated diabetes mellitus, increased corticosteroid excretion would favor the diagnosis of Cushing's syndrome.

With regard to the therapeutic implications, the hyperglycemic effects of cortisone therapy need only be watched for and controlled. The likelihood of retinal and renal vascular complications is not great but should be kept in mind when treating diabetic patients with cortisone. The value of ACTH or cortisone therapy in spontaneous hypoglycemia needs no further comment. Lastly, removal of the adrenals or pituitary in diabetics is still experimental therapy, yet to be proved to have definite value although it may offer some hope in the future handling of the vascular complications of diabetes mellitus.

SUMMARY

The relationship between diabetes mellitus and adrenocortical function has been presented. Experimental observations in normal and pancreatectomized animals as well as clinical observations in patients are reviewed, and the clinical implications of these findings are discussed.

REFERENCES

1. Jackson, W. P. U.: A concept of diabetes, Lancet 2:625 (Sept. 24) 1955.

2. Long, C. N. H., and Lukens, F. D. W.: The effects of adrenalectomy and hypophysectomy upon experimental diabetes, J. Exp. Med. 63:465 (April) 1936.

3. Long, C. N. H.; Katzin, B., and Fry, E. G.: The adrenal cortex and carbohydrate metabolism, Endocrinology 26:309 (Feb.) 1940.

4. Lukens, F. D. W.: Experimental diabetes and its relation to diabetes mellitus, Am. J. Med. 19:790 (Nov.) 1955.

5. Wick, A. N.; Drury, D. R., and MacKay, E. M.: The disposition of glucose by extrahepatic tissues, Ann. New York Acad. Sc. 54:684 (Dec. 28) 1951.

6. Ingle, D. J.: Some studies on the role of the adrenal cortex in organic metabolism, Ann. N. Y. Acad. Sc. 50:576 (July 27) 1949.

7. Ingle, D. J., and Thorn, G. W.: Comparison of effects of 11-desoxycorticosterone acetate and 17-hydroxy-11-dehydrocorticosterone in partially depancreatized rats, Am. J. Physiol. 132:670 (April) 1941.

8. Pincus, G., and Elmadjian, F.: The pituitary-adrenal system, Ann. Rev. Physiol., 16:403, 1954.

9. Houssay, B. A.: Carbohydrate metabolism, New

England J. Med. 214:971 (May 14) 1936.

10. Olmsted, J. M. D., and Logan, H. D.: Effect of insulin on the central nervous system and its relation to the pituitary body, Am. J. Physiol 66:437 (Oct.) 1923.

11. Young, F. G.: Permanent experimental diabetes produced by pituitary (anterior lobe) injections, Lancet 2:372 (Aug. 14) 1937.

12. Lukens, F. D. W., Dohan, F. C., and Wolcott, M. W.: Pituitary diabetes in the cat; recovery following phlorrhizin treatment, Endocrinology 32:475 (June) 1943.

13. Haist, R. E., Campbell, J., and Best, C. H.: Prevention of diabetes, New England J. Med. 223:607 (Oct. 17) 1940.

14. Young, F. G.: The growth hormone and diabetes, Recent Prog. Hormone Research 8:471, 1953.

15. Engel, F. L., Viau, A., Coggins, W., and Lynn, W. S., Jr.: Diabetogenic effect of growth hormone in the intact force-fed adrenocorticotrophin treated rat, Endocrinology 50:100 (Jan.) 1952.

16. deBodo, R. C., and Sinkoff, M. W.: The role of growth hormone in carbohydrate metabolism, Ann. New York Acad. Sc. 57:23 (June 30) 1953.

17. deBodo, R. C., and Sinkoff, M. W.: Anterior pituitary and adrenal hormones in the regulation of carbohydrate metabolism, Recent Prog. Hormone Research 8:511, 1953.

18. Venning, E. H., Kazmin, V., and Bell, J. C.: Biological assay of adrenal corticoids, Endocrinology 38:79 (Feb.) 1946.

19. Segaloff, A., and Many, A. S.: The role of adrenal steroids and ACTH in gluconeogenesis. A study in phloridzinized animals, Endocrinology 49:390 (Sept.) 1951.

20. Thorn, G. W., Renold, A. E., Morse, W. I., Goldfien, A., and Reddy, W. J.: Highly potent adrenal cortical steroids: Structure and biologic activity, Ann. Int. Med. 43:979 (Nov.) 1955.

21. Liddle, G. W., and Richard, J. E.: 2-methyl hydrocortisones: A new series of steroids with enhanced potency and prolonged action, Science 123:324 (Feb. 24) 1956.

22. Becker, B.: Diabetic retinopathy, Ann. Int. Med. 37:273 (Aug.) 1952.

23. Bloodworth, J. M. B., Jr., and Hamwi, G. J.: Histopathology of experimental glomerular lesions simulating human diabetic glomerulosclerosis, Am. J. Path. 31:167 (Jan.-Feb.) 1955.

24. Ricketts, H. T.: The problem of degenerative vascular disease in diabetes, Am. J. Med. 19:933 (Dec.) 1955.

25. Levine, R., and Goldstein, M. S.: On the mechanism of action of insulin, Recent Prog. Hormone Research 11:343, 1955.

26. Engle, F. L.: Editorial, The adrenal cortex and the metabolic response to stress, J. Clin. Endocrinol. & Metab. 13:1555 (Dec.) 1953.

27. Conn, J. W., Louis, L. H., and Wheeler, C. F.: Production of temporary diabetes mellitus in man with pituitary adrenocorticotropic hormone; relation to uric acid metabolism, J. Lab. & Clin. Med. 33:651 (June) 1948.

28. Sprague, R. G., Mason, H. L. and Power, M. H.: Physiologic effects of cortisone and ACTH in man, Recent Prog. Hormone Research 6:315, 1951.

29. Burns, T. W., Engle, F. L., Viau, A., Scott, J. L., Jr., Hollingsworth, D. R., and Werk, E.: Studies on the interdependent effects of stress and the adrenal cortex on carbohydrate metabolism in man, J. Clin. Invest. 32:781 (Aug.) 1953.

30. Persky, M., Linsk, J., Isaacs, M., Jenkins, J. P., Rosenbluth, M., and Kupperman, H. S.: Acute effects of intravenous hydrocortisone on glucose and insulin tolerance, and levels of serum and urinary inorganic phosphorus, J. Clin. Endocrinol. & Metab. 15:1247 (Oct.) 1955.

31. Kinsell, L. W., Michaels, G. D., Margen, S., Partridge, J. W., Boling, L., and Bolch, H. E.: The case for cortical steroid hormone acceleration of neoglucogenesis from fat in diabetic subjects. A summary of five years investigative work, J. Clin. Endocrinol. & Metab. 14:161 (Feb.) 1954.

32. Fajans, S. S., Louis, L. H., and Conn, J. W.: Metabolic effect of ACTH upon pre-existing diabetes mellitus, J. Clin. Endocrinol. 11:455 (May) 1951.

33. Conn, J. W., and Fajans, S. S.: Influence of adrenal cortical steroids on carbohydrate metabolism in man, Diabetes 5:114 (March) 1956.

34. Fajans, S. S., and Conn, J. W.: An approach to the prediction of diabetes mellitus by modification of the glucose tolerance test with cortisone, Diabetes 3:296 (July-Aug.) 1954.

35. McQuarrie, I.: Idiopathic spontaneously occurring hypoglycemia in infants, Am. J. Dis. Child. 87:389 (April) 1954.

36. Conn, J. W., and Seltzer, H. S.: Spontaneous hypoglycemia, Am. J. Med. 19:460 (Sept.) 1955.

37. Knowlton, A. I.: Cushing's syndrome, Bull. N. Y. Acad. Med. 29:441 (June) 1953.

38. Cope, O., and Raker, J. W.: Cushing's disease. The surgical experience in the care of 46 cases, New England J. Med. 253:119, 165-172 (July 28 and Aug 4) 1955.

39. Balfour, W. M., and Sprague, R. G.: Association of diabetes mellitus and disorders of the anterior pituitary, thyroid, and adrenal cortex, Am. J. Med. 7:596 (Nov.) 1949.

40. Davidoff, L. M., and Cushing, H.: Studies in acromegaly: disturbances of carbohydrate metabolism, Arch. Int. Med. 39:751 (June) 1927.

41. McCullagh, E. P., and Alivisatos, J. G.: Diabetes of anterior pituitary and adrenal cortical origin, Diabetes 3:349 (Sept.-Oct.) 1954.

42. Daughaday, W. H., Perry, W. E., and MacBride, C. M.: Hyperadrenalcorticism in a case of acromegaly with insulin resistant diabetes, J. Clin. Endocrinol. 10: 410 (April) 1950.

43. Martin, M. M., and Pond, M. H.: Pituitary insufficiency associated with diabetes mellitus, J. Clin. Endocrinol. & Metab. 14:1046 (Sept.) 1954.

44. Harvey, J. C., and deKlerk, J.: The Houssay phenomenon in man, Am. J. Med. 19:327 (Sept.) 1955.

45. Poulsen, J. F.: Recovery from retinopathy in a case of diabetes with Simmond's disease, Diabetes 2:7 (Jan.-Feb.) 1953.

46. Luft, R., Olivecrona, H., and Sjogren, B.: Hypophysectomy in man: experience in severe diabetes, J. Clin. Endocrinol. & Metab. 15:391 (April) 1955.

47. Kinsell, L. W., Lawrence, L., and Weyland, R. D.: Hypophysectomy in severe juvenile diabetes, J. Clin. Endocrinol. & Metab. 15:859 (July) 1955.

48. Thorn, G. W., Keopf, G. F., Lewis, R. A., and Olsen, E. F.: Carbohydrate metabolism in Addison's disease, J. Clin. Invest. 19:813 (Nov.) 1940.

49. Bergenstal, D. M., and Dao, T. L. Y.: Management of Addison's disease in adrenalectomized patients, Bull. New York Acad. Med. 29:295 (April) 1953.

50. Green, D. M., Nelson, J. N., Dodds, G. A., and Smalley, R. E.: Bilateral adrenalectomy in malignant hypertension and diabetes, J.A.M.A. 144:439 (Oct. 7) 1950.

51. Wortham, J. T., and Headstream, J. W.: Adrenalectomy in human diabetes. Effects in diabetics with advanced vascular disease, Diabetes 3:367 (Sept.-Oct.) 1954.

52. Venning, E. H., and Browne, J. S. L.: Excretion of glycogenic corticoids and of 17-ketosteroids in various endocrine and other disorders, J. Clin. Endocrinol. 7:79 (Feb.) 1947.

53. McArthur, J. W.; Sprague, R. G., and Mason, H. L.: The urinary excretion of corticosteroids in diabetic acidosis, J. Clin. Endocrinol. 10:307 (March) 1950.

54. Grinspoon, L., Sagild, U., Blum, H. P., and Marble, A.: Changes in circulating eosinophils in juvenile diabetics in responses to epinephrine, ACTH and hypoglycemia, J. Clin. Endocrinol. & Metab. 13:753 (July) 1953.

55. Freeman, S.; Wheeler, J. X., and Hoegemeier, H. W.: Free reducing and hydrocortisone-like steroids in human plasma, A.M.A. Arch. Int, Med. 97:45 (Jan.) 1956.

56. Walloch, S., Englert, E., Jr., and Brown, H.: Adrenocortical activity in diabetic acidosis, Clin. Res. Proc. 4:61 (Jan.) 1956.

57. Izzo, J. L., and Ellers, A: Effect of cortisone on 17-ketosteroid excretion in patients with diabetes mellitus, Science 123:417 (March 9) 1956.

DISCUSSION

Dr. William M. Luikart (Baton Rouge): Effects of adrenocortical function which are related to the problem of diabetes, and upon which there seems to be general agreement seem to me as follows:

Increased hepatic gluconeogenesis from protein.

Increased gluconeogenesis from fat.

Increased glycogenesis, of apparently greater degree in the liver, as compared to insulin's greater muscle glycogenesis.

Decreased insulin sensitivity either by altered cation concentration, or through stimulation of alpha cells glucagon production.

Tendency to foster capillary damage in diabetics.

There would seem to be no question as to the importance of the relation of adrenocortical function to the clinical syndrome of diabetes, if only from the fact that either hypophysectomy or adrenalectomy in certain respects reverse the biochemical state of the diabetic. The crux of the matter centers about the question of whether or not adrenal cortical function holds a merely permissive role in diabetes: The fact that carbohydrate metabolism in hypercorticism does appear to differ materially from the diabetic state lends support to the permissive role of the adrenal cortex. Collateral evidence is the suggestive reports indicating that the cortex plays a permissive role in the stress reaction, which is undoubtedly a factor in clinical diabetes.

Considering the very important effects of diabetes on the vasculature in relation to microaneurysmal changes in capillaries of retina or kidney, and especially to the well known acceleration of atherosclerotic changes, the problem of adrenocortical effects on lipid metabolism in diabetes deserves more study, such as is noted in reports by Harris and by Hirsch in separate papers on the association of hyperlipemia with the hyperglycemia of diabetes. If insulin does promote lipogenesis, and adrenocortical function indeed promotes gluconeogenesis from fat, an important relationship is strongly suggested. As has been pointed out in the present paper, adrenalectomy in certain cases has brought about amelioration of vascular degeneration in diabetics.

It seems clear that the anterior pituitary exerts some of its effects on diabetes through the adrenal cortex, but also has some direct effects. Of particular interest is the apparent demonstration of antagonistic effects between pituitary growth hormone and adrenocortical hormone, especially since insulin itself has been shown to act as a growth hormone under some conditions, and, in addition, insulin appears to be required for the growth-promoting activity of pituitary growth hormone.

The specific points of action of insulin and adrenal cortical hormone are of prime interest. While the work and theories of the Coris and of Stadie can scarcely be dismissed, the theory of insulin action presented by Levine is most interesting. The demonstration that the insulin resistance induced by cortisone could be prevented if potassium and sodium concentrations were maintained at normal levels, would suggest the possibility that the effect of insulin on membrane permeability to glucose is somehow related to cation concentration, which in turn is known to be affected by adrenal cortical hormone; this might represent a point at which insulin and adrenocortical hormone are specifically antagonistic.

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RECONSTRUCTION PROCEDURES OF THE HIP *

T. E. BANKS, JR., M. D.
ALEXANDRIA

The term reconstruction can be interpreted to mean to "re-create". This recreation can be thought of in two different ways: (1) to restore the anatomy of a diseased hip joint, or (2) to restore the function of such a joint. The purpose of this paper is to summarize in a rather general manner the procedures which have been designed over the past forty to fifty years and to present the basic principles of some of these procedures with their results. No attempt will be made to evaluate these procedures critically. Also, the use of femoral head prostheses will not be discussed.

The types of cases with which we are dealing have been "problem cases". They include for the most part congenital dislocations of the hip, osteoarthritis of the hip with pain and varying degrees of ankylosis, nonunion following fracture of the neck of the femur with and without aseptic necrosis of the femoral head, and associated cases. A reconstruction operation should have as its object; (1) the restoration of stability and (2) the preservation of motion. To quote Royal Whitman, "In the treatment of fractures, the first indication is to save life, the second to get

* Presented at the Seventy-sixth Annual Meeting of the Louisiana State Medical Society, Alexandria, April 24, 1956.

union, and the third to correct or diminish displacements".

It is difficult to separate the true (anatomically) reconstructive procedures from the functionally reconstructive procedures (osteotomies), except on an historical basis. Even at the present time a decision for a procedure may be changed during an operative procedure due to findings unexpected prior to surgery. For this reason, it behooves us to be conversant with the variety of procedures which have been recorded.

In 1917, Brackett [1] reported a procedure in nonunions of the femoral neck which consisted of removal of the top of the greater trochanter and the placing of the femoral head fragment on the supermedial aspect of the trochanter. Muscles were then attached to the lateral side of the femur and plaster immobilization was used. He reported an eight-year follow-up on a case in 1925 [2] with apparently satisfactory result.

Albee's original description was presented in 1919, but in 1929,[3] he presented a summary of 110 cases, in which they were graded as results of 87 excellent, 14 fair, 9 poor, and 2 deaths. His procedure consisted of removal of the femoral head, placing of the neck into the acetabulum, splitting the trochanter in a sagittal plane, and placing autogenous iliac bone between the cut surfaces of the trochanteric fragments to keep the mechanical advantage of the abductor muscles. Complete immobilization in plaster was necessary for eight to ten weeks following surgery.

Royal Whitman,[4] in 1921, presented what he classified as a true reconstructive operation. In it, he removed the femoral head, placed the neck and shaft into the acetabulum, and transplanted the trochanter with its attached muscles inferiorly to the lateral side of the femoral shaft. The patient was then immobilized in plaster for eight to ten weeks. He felt that his procedure was more mechanically sound than Albee's, as it did not shorten the excursion of the abductor muscles.

The relative merits of these two procedures suffered a controversy for many years. Speed, in 1926,[5] felt that the Brackett procedure had the complication of instability in the hip and felt the Whitman procedure had given the best results. Magnuson, in 1932,[6] preferred the Brackett procedure to the Whitman, depending on the condition of the head fragment. At the same time he advocated a procedure which combined the two principles—with retention of the head fragment, shaping of the neck remnant and placing it into the head, and transplantation of the trochanter.

Colonna, in 1935,[7] designed a procedure primarily for those cases in which nonunion is accompanied by complete absorption of the femoral neck. In his procedure all of the muscles are removed from the trochanter, *but* the trochanter is left covered with a smooth fibromuscular layer. The head fragment is removed and trochanter is placed in the acetabulum. The abductor muscles are reattached on the lateral side of the femur and the vastus lateralis is reefed over the muscle attachment. Here again, plaster immobilization for four weeks followed by overhead suspension for three to four weeks is necessary.

Luck, in 1938,[8] varied the reconstruction ideas a bit with his procedure designed to eliminate shearing forces and to prepare a vascular bed in the intertrochanteric region for the fractured surface of the femoral head. If the head was not dead and not arthritic, it was used; otherwise, a Colonna was done, or a Whitman. The procedure consisted of the resection of a wedge from the medial aspect of the shaft and neck at the level of the lesser trochanter, placement of the head atop the raw shaft of the femur, and transplantation of the trochanter inferiorly along the lateral side of the femur. This was followed by plaster immobilization in 20 degrees of abduction for about eight weeks.

Heretofore, these procedures were designed primarily for nonunions of the femoral neck. About this time in medical history the use of osteotomies in this area began to gain popularity. Blount [9] gives credit to Kirmisson, in 1894, for report

of first osteotomies in the subtrochanteric area for gait correction in 1918, and Lorenz in 1919, as initial advocates of corrective osteotomy.

A great deal of thanks and credit is due McMurray for his widespread use of the osteotomy. In his original article in 1936,[10] he advocated a high oblique osteotomy which eliminated the shearing forces at the site of femoral neck injury. His paper, in 1938,[11] was the first to advocate this procedure in fresh transcervical fractures. His essentials for good rusults were listed as (1) correct position for transferred shaft of the femur, (2) prolonged adequate postoperative immobilization, and (3) fixation of the limb in very slight abduction or in neutral to prevent knock-knee deformity. McMurray was also the first to advocate traction preoperatively in cases with upward displacement of the trochanter.

From that time to the present the progress has been made primarily in the direction of osteotomies for functional reconstruction of the hip. Gaenslen, in 1935,[12] preferred the transverse osteotomy of Schanz in preference to the oblique osteotomy of McMurray, as the former does not depend on ischial abutment. He felt that either the high or low osteotomy could be used for old ununited hip fractures, but the low only in congenital dislocations of the hip.

Blount, in 1943,[13] marked another milestone of progress when he made an effort to eliminate postoperative cast immobilization. He designed a double angle blade plate to be used with what is basically a high or low Schanz osteotomy with internal fixation.

Dickson, in 1947,[14] designed what is referred to as his geometric osteotomy. He did not agree with the extensive dissection necessary for the Brackett procedure, as he felt it interfered with circulation. He devised an osteotomy in the subtrochanteric region hexagonal in shape with fixation with a single angle blade plate. By rotation of the head and neck fragments 60 degrees he desired to convert the neck fracture to a transverse one. He now advocates the adduction of the proximal fragment 45 degrees instead of 60 degrees.

Blount[9] summarized, in 1952, an extremely useful and complete basis for the use of osteotomy both in the osteoarthritic hip and in the femoral neck fractures, old and new. Basically, he feels that all osteotomies can be divided into either angulation or displacement osteotomies.

Angulation osteotomy is used when better mechanical function of the hip joint is desired. In fractures of the neck of the femur it is used when the head is alive and when union across the fracture site is expected. It is also used in congenital dislocations to correct adduction and faulty rotation when no healing of a fracture site is necessary. In angulation osteotomy there must be two fixation devices, one to hold the osteotomy site and the other to fix the head fragment.

Displacement osteotomy is peculiarly suited to degenerative arthrosis of the hip, ununited fracture with avascular necrosis of the femoral head, and occasional cases of tuberculosis. By this procedure the operator is trying to relieve the femoral head from weight bearing.

Internal fixation can now be used to prevent the need for postoperative cast immobilization.

Simultaneous obturator neurectomy is done by some operators routinely at the time of displacement osteotomy to relieve pain due to impingement of muscles at the time of osteotomy.

For degenerative arthrosis the displacement osteotomy is more popular in countries other than the United States, but with the use of internal fixation, it is gaining popularity.

Milch first reported, in 1943,[15] the use of resection of the femoral head with pelvic support osteotomy for ankylosis of the hip. He summarized a follow-up on cases with some more detailed advantageous changes in technique in a report in 1955.[16] He shows that femoral mobility is produced by resection of the femoral head and neck. Re-establishment of stability is by means of a pelvic-support osteotomy. The post-

osteotomy angle must be congruent with the angle of inclination of the pelvic wall. This varies from 206 degrees in the female to 211 degrees more or less percentage of error, and it must be determined preoperatively.

We have not discussed today the use of arthrodesis in the treatment of the same conditions. With particular reference to unilateral hip joint disease, we do not forget the frequent use of arthrodesis to afford pain-free stability of a diseased hip joint.

SUMMARY

A brief review of some of the basic principles of reconstructive procedures of the hip has been presented—along with some attempt to tabulate the advances. The age-old problems of hip joint disease and injury have not all been solved, but with the strides in internal fixation, in lessening postoperative immobilization, and in the stress on functional reconstruction, we feel that we have something to offer a higher percentage of these patients. Osteotomy is not to be advocated in every case, but when it is applicable it is frequently the simplest solution to the problem and the one most likely to endure. The uses of arthrodesis or arthroplasty must not be overlooked in our evaluation of cases. Arthroplasty, prosthetic femoral heads, and as yet some undiscovered procedures will always be preferable in certain cases, but they should not be used to the exclusion of the simpler and frequently more effective osteotomies.

REFERENCES

1. Brackett, E. G. and New, M. S.: Treatment of old ununited fractures of the neck of the femur by transplantation of the head of the femur to the trochanter, Boston Med. & Surg. Jour. 177: 351, 1917.

2. Brackett, E. G.: Fractured neck of femur: operation of transplantation of femoral head to trochanter, Boston Med. & Surg. Jour. 192: 1118, 1925.

3. Albee, Fred H.: Treatment of ununited fracture of the neck of the femur, Surg. Gynec. & Obst. 49: 810, 1929.

4. Whitman, Royal: Reconstruction operation for ununited fracture of the neck of the femur, Surg. Gynec. & Obst. 32: 479, 1921.

5. Speed, J. S.: Reconstruction operation on the hip, J.A.M.A. 87: 1631, 1926.

6. Magnuson, P. B.: The repair of ununited fracture of the neck of the femur, J.A.M.A. 98: 1791, 1932.

7. Colonna, Paul C.: A new type of reconstruction operation for old ununited fracture of the neck of the femur, J. Bone & Joint Surg. 17: 110, 1935.

8. Luck, Vernon J.: Reconstruction operation for psudoarthrosis and resorption of neck of femur, J. Iowa Med. Soc. 28: 620, 1938.

9. Blount, Walter P.: Proximal osteotomies of the femur, The A.A.O.S. Instructional Course Lectures IX: 1, 1952.

10. Mc Murray, T. P.: Ununited fractures of the neck of the femur, J. Bone & Joint Surg. 18: 319, 1936.

11. Mc Murray, T. P.: Fracture of the neck of the femur treated by oblique osteotomy, British M. J. 1: 330, 1938.

12. Gaenslen, F. J.: The Schanz subtrochanteric osteotomy for irreducible dislocation of the hip, J. Bone & Joint Surg. 17: 76, 1935.

13. Blount, Walter P.: Blade-plate internal fixation for high femoral osteotomies, J. Bone & Joint Surg. 25: 319, 1943.

14. Dickson, James A.: High geometric osteotomy with rotation and bone graft for ununited fractures of the neck of the femur, J. Bone & Joint Surg., 29: 1005, 1947.

15. Milch, Henry: Resection of the femoral neck with pelvic support osteotomy for ankylosis of the hip, Surgery 13: 55, 1943.

16. Milch, Henry: The resection-angulation operation for hip-joint disabilities, J. Bone & Joint Surg. 37-A, No. 4: 699, 1955.

SOME ASPECTS OF ALCOHOLISM *

KENNETH A. RITTER, M. D. †
NEW ORLEANS

I would like to discuss two aspects of the character disorder known as chronic alcoholism, one dealing with understanding of the behavior pattern, and the other concerning the dynamics of effective treatment.

For centuries, alcoholism has been a mysterious malady, particularly baffling and frustrating to the physician called upon to treat it. In the past few decades, the veil of mystery has been slowly pushed aside so that today many of us have entirely new attitudes about it.

With the introduction into psychiatry of the idea that all behavior, from the average to the most bizarre, has an understandable purpose, and that we need only keep our eyes open for these purposes to become apparent, it becomes possible to throw light into previously darkened areas. As it later developed, it was necessary to survey the entire life history of an individual in order to see clearly why he behaved in one way or another. Keeping this concept in mind, I believe we can begin to understand alcoholic behavior. As I have implied, it is necessary to com-

* Presented at the Seventy-sixth Annual Meeting of the Louisiana State Medical Society, Alexandria, La., April 24, 1956.

† Instructor in Psychiatry, LSU School of Medicine, New Orleans.

prehension that we go beyond investigation of just the symptoms and see the patient in his past and present environments, when he is sick and when he is well.

We can arbitrarily begin by observation of a representative sample while he is sober. He impresses us as a reserved, sensitive, shy person, extraordinarily contented with passive activities. He is usually of average, and sometimes of superior intelligence, has often held good jobs in which he may have excelled but rarely is he promoted and seldom is he self-employed. In a social setting without alcohol, although usually acceptable, he rarely expresses his opinions, preferring simply to agree with those of others. If he does express an opinion, it is readily changed by the pressure of persuasion. He has a constricted range of interest enjoying no constant hobbies or extracurricular activities. To many, he is "a nice guy", and you know this defines people who always agree with us. He rarely gets angry, even when provoked. If married, he "gets along" with his wife, but she seems always to be the more aggressive, more opinionated, "stick - to - your - guns" type than he. If single, his interest in the opposite sex is often fleeting and inconsistent. He is ordinarily in debt, sometimes heavily so.

Now what happens when he begins to drink? This ordinarily quiet, reserved, nice guy becomes increasingly verbose and opinionated, more resistive to persuasion, more assertive, and is now easily angered, sometimes he even fights. He appears more comfortable and is a match for anyone in an argument. There seems to be more than the usual degree of personality change with the addition of the spirits. Later, though, when everyone else has gone home, he remains, still drinking, consuming more than anyone else, and this may go on for several days, weeks, or months. His family suffers, his employer suffers, his anxious friends suffer, his bank account and debtors suffer. He causes pain and anguish to everyone concerned with him. Finally, he is exhausted and delirious and passes through the withdrawal ordeal, often in jail or serving a sentence in the local workhouse. He arrives at sobriety to receive the exhortations, warnings, threats, pleas, advice and sermons of the well-meaning aforementioned, designed to stir up his guilt for this "shameful" behavior, and to make him sufficiently repentent and contrite, and he again obligingly agrees. Sooner or later this series of events is repeated, and he eventually incurs the frustration-procured wrath and rejection of all. He may in time slip to the "skid row" level, working only to keep himself supplied with the soporific ego remedy. Reliability, honesty, ability to respect himself are no longer his. Thus the all-too-familiar pattern of alcoholic behavior.

As doctors we usually see and follow the progress of the disease only from the time the symptoms become overt and troublesome, (more often after they have become troublesome to others). The pathology, however, has been present for a long time, and the incubation period here is often in terms of *years*. I suspect that all *true* alcoholics experience similar emotional events which begin very early in life, and continue on through their formative years.

What is this common experience the alcoholics go through in their early life? The significant problem they all have is one with which you are familiar—maternal domination. There seems to be a need in these mothers to surround themselves with the helpless, which includes their husbands, probably in order to feel strong themselves. Such mothers make the helpless attitude too attractive and discourage growing up. Therefore, the child never gets over the attachment to this period in his life when his needs are sensed and fulfilled immediately; mother does not even wait for him to cry to show that he needs something; she sees to it that he always gets a lot more than he needs, and he experiences little or no denial. Later we find that such careful training has created a placid

child, very agreeable, and easily bent to the will. He comes to expect and rely on her opinions, her judgment, and if he differs, he learns that he must not "sass" or else she may remove entirely the expected support. He secretly yearns to be independent, to get on his own, but she refuses to allow this, preferring her "wisdom" to his, and never permitting him the pleasure of making mistakes. Whatever individualism he has must be suppressed and he grows up and up, ever in the ominous shadow of mother's skirts, all the while storing up a lot of angry feelings over being denied the right to take his chances with the rest. So the boy grows into the man, but he is a crippled man, unaccustomed to the adult prerogatives of making mistakes, asserting himself, making decisions and sticking by them, taking the blame and being deprived.

Let me say here that there are degrees of crippling, just as in other pathological conditions, and I am sure, under the proper circumstances ("corrective emotional experiences") a certain amount of healing can take place. This may account for some variation in resistance to the disease.

When faced with these adult privileges, he becomes confused and frightened at these unknown things, and begins to long for the secure feeling he knew when mama ruled the roost. He unconsciously and automatically expects of others that they should treat him like she did; he unconsciously perceives the world as peopled with "Big Mamas". A lot of inner conflict develops because others do not treat him in this manner, and he has learned no other way to relate to them. This produces an ever increasing amount of painful feelings and anger.

Then, like manna from heaven, comes his discovery of alcohol. With this he finds that he can get the pleasurable, temporary feeling of maturity, that he can acquire the feeling of "I can" and temporarily abolish the feeling of "I can't" stored somewhere in his cortex. Later he discovers that he can at last express his anger and defiance, and can retaliate against his mother and her surrogates (society) by making them suffer through his prolonged drinking. This is his way of "winning the battles" and proving that he is master of his own destiny. All the while he is operating on the unconscious delusion that he is gaining mastery over mama. One can see that he is attempting to return to his earlier years, a regression which demonstrates beautifully the conflict between maturity and immaturity.

A simple way of summarizing the entire matter would be to say that the sober period represents the desire to be "good" (to be passive, completely agreeable with his strong mother-conscience) and the drinking period symbolizes the desire to be "bad" (to be mature, aggressive, opinionated, decisive, manly.)

Now to consider treatment. Since we know what the alcoholic is striving for, we can more effectively deal with his problem. His behavior constantly provokes us to act like the rest, to reject and condemn him, to advise, and coerce. But this is fatal to success in treatment. The therapist's job is to take an attitude quite *opposite* to the one expected. Now this is not as simple as it sounds. He calls from a jail or bar at all hours of the night, fails to pay his bill, misses his appointments, or keeps them, but presents himself in the office "stewed to the gills". Such activities make it very difficult, to say the least, in maintaining a kindly sympathetic permissive attitude. So often this acting-out "protects" the patient from effective individual therapy by inciting the rejection of the psychiatrist.

A.A. treatment seems to produce better results in the average alcoholic than individual psychotherapy, and these, I believe are the reasons why. This group believes in a physical and moral cause for alcoholism, thereby removing the stigma of psychiatric illness, and making treatment by them less threatening. They are really the only eclectics since they believe in these causes, yet certainly use psychi-

atric treatment on each other. The patient gets nowhere with his provocative behavior since they charge no fees, make no demands, only offer their help, are available day or night, and are tolerable and sympathetic and devoted always. What is a liability in average society becomes an asset in this new society—You *must* be an alcoholic to gain admittance to this exclusive club. The patient learns that shame and guilt is not warranted here, even competing with others in telling of his past "binges", and eventually becoming very proud that he was once an alcoholic; so much so, that he often remains anything but anonymous. Another important factor in operation, of course, is his identification with the "strong" and the pride in helping the "weak". Without question, the success of any form of treatment depends on how effective one is in making the sober period more pleasant, tolerable and attractive, and in eliminating the shame, guilt and remorse of the drinking period.

I have tried to summarize here some of my ideas as to the dynamics in the disease process and repair process in chronic alcoholism I must admit that my experience in this field has been limited to a certain class of patients and that what I described may not apply to the entire group. It is my hope that these concepts may be stimulating to you.

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TYPHOID FEVER - RICHLAND PARISH 1954-1955

J. D. ORGERON, M. P. H.*
CHARLES T. CARAWAY, D. V. M.*
ROSE MARY MARTINE, R. N.*
J. D. MARTIN, M. D.*

Two outbreaks of typhoid fever occurred in Richland Parish, Louisiana, in 1954 - 1955. One outbreak started in Arkansas, spread to Louisiana, and from there to California. The other started in and was confined to Richland Parish, Louisiana. Twenty-nine residents of Richland Parish became infected and 4 associated cases were residents of other states (Arkansas 2, and California 2).

Richland Parish has a population of about 29,000 persons, 60 per cent of whom are white.

The area of the parish in which the outbreak occurred is made up largely of cotton farms operated by Negro tenants. These families live in small frame homes under crowded conditions. Their personal and household hygiene habits are very poor. Few of the farms possess a safe water supply, sanitary excreta disposal, or other sanitary facilities.

Typhoid is endemic in Richland Parish. In the period 1946 through 1955, an average of 7 cases per year were reported. Physician reports of communicable diseases occurring in rural areas rarely show the exact place of residence of the sick person. The exact number of cases of typhoid occurring in previous years and residing in the locale presently affected therefore is unknown.

Typhoid, in recent years in Louisiana, has occurred in sporadic outbreaks. Widespread explosive epidemics due to contaminated milk and water supplies are no longer observed. Most cases today are the result of contact with a recent case of typhoid or with a typhoid carrier.

In May 1954, 5 cases of typhoid fever were diagnosed in Richland Parish and reported to the parish health unit. Epidemiological investigations were started by the personnel of the parish health unit, but as the number of reported cases increased, assistance was requested of the State Department of Health.

The first diagnosed case was an 18 year old Negro, female from El Dorado, Ark. Unfortunately, the Vi bacteriophage type of the typhoid organism causing this woman's illness was never determined. She became ill on March 13, 1954. This patient's parents live in Richland Parish and she gives a history of frequent visits to their household.

Louisiana's first case was the 13 year old sister of the Arkansas woman. This child visited her sister in Arkansas on April 4, 1954, at which time the Arkansas

* Section of Epidemiology, Louisiana State Department of Health.

woman was hospitalized with typhoid. This Louisiana child became ill on April 7, 1954. Since only three days elapsed between the date of the visit and the date of onset, it seems unlikely that she contracted her infection while in Arkansas.

Extensive investigation of this, the first Louisiana case and her contacts, revealed contact with the 2 year old son of the Arkansas woman and that this child had had a intermittent diarrheal disease for about sixteen months. *Salmonella typhosa*, bacteriophage type J was isolated from his stool. At the time of this isolation, *Salmonella typhosa* bacteriophage type J had not been previously isolated from typhoid cases in Louisiana.

The child of the Arkansas woman spent considerable time at the home of his grandparents in Louisiana, but exact dates are not available. His mother states that he had had recurrent bouts of diarrhea since February 1953, and had been treated with various drugs with varying degrees of success. This child remained in an affected household in Louisiana during the outbreak but at no time did he develop clinically recognized typhoid.

Subsequent Louisiana cases were reported in members of households nearby the residence of the 13 year old girl. Seven cases occurred during the first eleven days of 1955. This was probably the result of increased associations between members of infected and non-infected households during the Christmas season.

By April 1955, the outbreak appeared to be under control as there had been no reported cases in the preceding three-month period. In May, however, a 42 year old Negro, female developed typhoid. This case was believed to be an extension of the original outbreak as she gave a history of contact with members of previously affected households. Two weeks after the onset of her illness, her husband was hospitalized with typhoid due to *Salmonella typhosa* bacteriophage type E-1. Since all previous infections were due to *S. typhosa* bacteriophage type J, we therefore realized that two outbreaks of typhoid were occurring at the same time. Five cases occurred in the type E-1 outbreak.

During investigation of the bacteriophage type E-1 cases, it was discovered that this family obtained raw milk from a neighbor. The neighbor denied ever having had typhoid but *S. typhosa* bacteriophage type E-1 has been isolated consistently from her stool. She is a chronic typhoid carrier.

Laboratory confirmation of the clinical diagnosis was obtained by isolation and identification of *Salmonella typhosa* in 18 of the 29 reported cases in Louisiana. The remaining 11 cases were accepted on the basis of clinical symptoms, strong epidemiological evidence and presumptive serologic findings.

Prior to these two outbreaks, there were no known typhoid carriers in Richland Parish.

Routine follow-up in Louisiana of confirmed typhoid cases consists of the cultural examination for *S. typhosa* of three stool and three urine specimens taken twenty-four hours apart at one, three, six, and twelve months after the discontinuance of drug therapy. If *S. typhosa* is isolated from either the stool or urine one year post treatment, the person is considered a typhoid carrier.

Three persons infected during this outbreak became *Salmonella typhosa* bacteriophage type J carriers and the woman who owned the milk cow has been established as a *Salmonella typhosa* bacteriophage type E-1 carrier.

Fourteen of the persons infected during the outbreak have been shown not to be carriers, 1 person, in addition to the 4 mentioned earlier, is strongly suspected of being a carrier and 11 other persons are under public health surveillance for determination of carrier status.

An analysis of the typhoid vaccination status of the 29 Louisiana cases merits comment. Seven persons were vaccinated less than one month before onset of disease; 2, one to six months before onset; 8, seven to twelve months before onset and 2, two years before onset. One of these last 2 had

received a booster inoculation one year before onset of illness. The remaining 10 cases could not definitely recall having completed a series of typhoid vaccine inoculations and therefore were considered as inadequately immunized.

The mode of spread in the *Salmonella typhosa* bacteriophage type J outbreak was probably through person to person contact. This belief is supported by the close proximity of the households, the poor personal hygiene of the members of the group, the lack of sanitary facilities, the frequent intermingling of the members of the groups and the chronological distribution of the onsets of the illness and because there were no common food or drink sources.

The *Salmonella typhosa* bacteriophage type E-1 outbreak resulted from the drinking of raw milk contaminated by a typhoid carrier.

The importance of good personal hygiene, a safe environment and typhoid vaccination was stressed repeatedly. New sanitary toilet facilities have been constructed, doors and windows screened, water supplies improved and fly control measures instituted at most of the households.

There is a need for further and continued education of this social group. Since sanitary excreta disposal and adequate handwashing facilities are not available, a potential threat to the health of the community exists at the local school attended by the 2 children who have become typhoid carriers.

SUMMARY

Two separate outbreaks of typhoid fever in Richland Parish were investigated and differentiated by the use of the Vi bacteriophage method of typing *Salmonella typhosa*. The first outbreak was spread by person to person contact, whereas the second outbreak resulted from the drinking of contaminated raw milk provided to the affected members of the household by a neighbor who was a previously unknown typhoid carrier. Twenty-eight cases (Louisiana 24, California 2, Arkansas 2) occurred in the J type outbreak and 5 in the E-1 outbreak. Richland Parish, which prior to 1954 had no known typhoid carriers, now has 4 known typhoid carriers.

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The Journal of the Louisiana State Medical Society

Established 1844

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SUBSCRIPTION TERMS: $4.00 per year in advance, postage paid, for the United States; $4.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

The Journal *does not hold itself responsible for statements made by any contributor.*

PHYSICIANS AND THE "MEDICARE" PROGRAM

Public Law No. 569 of the 84th Congress, known as Dependents' Medical Care Act, undertakes to provide medical care for some of the dependents of armed forces personnel through the use of civilian practicing physicians and facilities, in addition to that available from the armed forces, in such areas where care may be needed. The law goes into effect on December 8, 1956, having been passed and signed six months before. This law puts into operation a principle which has been supported by various laws of the Congress and by custom in military installations for over one hundred and forty years that the dependents of armed forces personnel are to be provided with medical care at government expense. The program in some respects resembles the E.M.I.C. program operating during World War II, but with provisions drawn in such a manner as to provide what is hoped to be a more satisfactory result.

The impact of the present "Medicare" Program on the physician will not be great at present, but the potentialities of this plan and the possibility of grafting state medicine on to it in the future are most important. An important effect will be to relieve, in some measure, the situation which calls for drafting of physicians into the armed forces to care for the dependents of armed forces personnel. To this extent it will contribute, it is hoped, to the ultimate defeat of the Doctor Draft law, which in spite of Congressional action and judicial support is obviously unjust and should be held unconstitutional. Another effect of this law will be to transfer care of some of the dependents of armed forces personnel from government agencies to that of organized medicine, which is of course into the private practice of medicine. A third, and also beneficial effect of the law, is the establishment of a definite policy on the part of the government for medical care of the dependents of the armed forces.

Representatives of the Defense Department have made strenuous and commendable efforts to cooperate with the physicians in implementing this law. Tentative plans have been put forward for discussion and clarification, conferences arranged with representatives of organized medicine, and our spokesmen have been equally diligent in their effort to produce a system that will provide satisfactory medical care. It is anticipated that the plan will provide temporary medical care at government expense for some 800,000 wives, children, and other dependents of

the armed forces personnel in this country. The armed forces representatives ask for a contracting agent who will supply the medical care and this is the state medical society. They also ask for a fiscal agent who will undertake to provide administrative clerical needs and serve as disbursing agent. In thirty-two of the states this will be Blue Cross agencies, and in sixteen states by private insurance carriers. In our state the Continental Service Life and Health Insurance Company of Baton Rouge will serve as our fiscal agent. This is the company which has taken over the responsibilities and serviced so efficiently and creditably the contracts of the former Louisiana Physicians Service Insurance Company, or Louisiana Blue Shield, when its difficulties reached the point of requiring outside help.

The law provides that dependents of the armed forces personnel will be cared for in the medical facilities of the uniformed services when these are available, provided this does not interfere with the effective utilization of the facilities for their primary purpose. The care would provide diagnosis, treatment of acute medical and surgical conditions, treatment of contagious diseases, immunization, maternity and infant care, but not for mental and nervous diseases, chronic diseases, or elective medical and surgical conditions except by special arrangement. Home calls are not required except in special cases.

The plan provides for the payment by the patient of hospital expenses up to $25, the government providing payments for amounts over this. The law also provides for a review, and if necessary, adjustments of payments by the armed forces under this plan, not later than one hundred and twenty days after the first year the plan is in effect and each year thereafter. In the many conferences that have been held in preparation for these arrangements it was emphasized that payments would be on a realistic and practical basis. A uniform fee schedule for the whole nation was regarded as impractical by our spokesmen. Each state society organization is in the process of furnishing the representatives of the armed forces with a fee schedule recommended for a particular state. Hospital charges are to be handled on a somewhat similar basis.

The involved processes of furnishing medical care for the government will be a tax on the time and patience of the physician. Physicians have the capacity to cooperate in the operation of this comprehensive medical plan. It is clearly to the very great interest of organized medicine and the individual physician to see that the plan works. The armed forces have it in their power under the laws to draft physicians for this work, to contract with agencies not under the direction of organized medicine, and to deal with organized medicine. We, as physicians, have reason to object to the first two of these alternatives. It behooves us to see that it works and is made satisfactory to the public, the armed forces, and ourselves.

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The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

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LIAISON COMMITTEE — PUBLIC POLICY & LEGISLATION AND CONGRESSIONAL COMMITTEES

H. W. Richmond, M. D., Oakdale; E. L. Zander, M. D., New Orleans.

LOUISIANA ORGANIZATIONS FOR STATE LEGISLATION

M. E. Kopfler, M. D., Chairman; Gordon W. Peek, M. D., Vice Chairman; Wiley A. Dial, M. D.; all of Baton Rouge; M. C. Wiginton, M. D., Hammond; Leo J. Kerne, M. D., Thibodaux; B. E. Trichel, M. D., Shreveport; J. F. Gladney, M. D., Homer; E. L. Carroll, M. D., Columbia; H. W. Richmond, M. D., Oakdale; J. T. Willis, M. D., Alexandria; Rafael C. Sanchez, M. D.; C. Grenes Cole, M. D.; both of New Orleans.

MEDIATION

C. J. Brown, M. D., Chairman; Morell W. Miller, M. D., H. T. Beacham, M. D.; all of New Orleans; M. E. Kopfler, M. D., Baton Rouge.

MEDICAL AND HOSPITAL SERVICES IN RE INSURANCE CONTRACTS

O. B. Owens, M. D., Chairman, Alexandria; Albert J. Ochsner II, M. D., Alexandria; Rhett McMahon, M. D., Baton Rouge; Jerome J. Romagosa, M. D., Lafayette; Paul D. Abramson, M. D. (Pres. LSMS), Shreveport; Ralph M. Hartwell, M. D.; P. H. Jones, M. D.; C. Grenes Cole, M. D. (Sec.-Treas. LSMS); all of New Orleans.

MEDICAL INDIGENCY

H. H. Hardy, Jr., M. D., Chairman, Alexandria; Paul M. Jackson, M. D., Clinton; Marion LeDoux, M. D., P. M. Tiller, M. D.; both of New Orleans.

MENTAL HEALTH

Gene Usdin, M. D., Chairman; F. W. Brewer, M. D.; both of New Orleans; D. H. Duncan, M. D., Shreveport.

NATIONAL EMERGENCY MEDICAL SERVICE (CIVIL DEFENSE)

Moss M. Bannerman, M. D., Chairman; Charles Mosely, M. D.; both of Baton Rouge; Henson S. Coon, M. D., Monroe; W. A. Ellender, M. D., Houma; J. A. Hendrick, Jr., M. D., Shreveport; Charles B. Odom, M. D., E. J. Joubert, Jr., M. D.; both of New Orleans; S. F. Fraser, M. D., Many; E. P. Breaux, M. D., Lafayette; F. C. Shute, Jr., M. D., Opelousas; H. H. Hardy, Jr., M. D., Alexandria.

NEUROPSYCHIATRIC SERVICE AT CHARITY HOSPITALS

Edmond Connely, M. D., Chairman; P. H. Jones, M. D., both of New Orleans; E. M. Robards, M. D., Jackson; Arthur L. Seale, M. D., Pineville.

NOMINATIONS (COMMITTEES TO BE ELECTED AT 1957 ANNUAL MEETING)

E. L. Leckert, M. D., Chairman, New Orleans; C. M. Horton, M. D., Franklin; Ralph H. Riggs, M. D., Shreveport.

PUBLIC HEALTH OF THE STATE OF LOUISIANA

B. J. DeLaureal, M. D., Chairman; W. P. Gardiner, M. D.; both of New Orleans; E. C. Faulk, M. D., Rayne; B. E. Spencer, M. D., Sterlington; Reed A. Fontenot, M. D., Ville Platte; Fred Mayer, M. D., Opelousas; G. Vasquez, M. D., Lake Charles; James Welch, M. D., Alexandria.

RURAL AND URBAN HEALTH

J. P. Saunders, M. D., Chairman, Shreveport; D. B. Barber, M. D., Alexandria; V. Blandino, M. D., New Orleans; E. L. Carroll, M. D., Columbia; Guy Jones, M. D., Lockport; B. M. Woodard, M. D., Lake Charles; M. C. Wiginton, M. D., Hammond.

STATE HOSPITAL POLICIES

George Wright, M. D., Chairman, Monroe; W. E. Barker, Jr., M. D., Plaquemine; T. A. Kimbrough, M. D., Lafayette; P. H. Jones, M. D., Charles B. Odom, M. D., Edwin L. Zander, M. D.; all of New Orleans; B. E. Trichel, M. D., Shreveport.

WOMAN'S AUXILIARY (ADVISORY COMMITTEE)

Walter Moss, M. D., Chairman, Lake Charles; W. A. K. Seale, M. D., Sulphur; E. L. Zander, M. D., C. Grenes Cole, M. D.; both of New Orleans; Paul D. Abramson, M. D. (Pres LSMS) ex-officio, Shreveport.

CHAIRMEN OF SCIENTIFIC SECTIONS 1957 ANNUAL MEETING

ALLERGY

Dr. Vincent Derbes, New Orleans

BACTERIOLOGY & PATHOLOGY

Dr. S. D. Cummins, Shreveport

CHEST

Dr. L. H. Strug, New Orleans

DERMATOLOGY

Dr. J. D. Youman, Jr., Shreveport

DIABETES

Dr. A. A. Herold, Sr., Shreveport

EAR, NOSE & THROAT

Dr. Jack Pou, Shreveport

EYE

Dr. L. F. Gray, Shreveport

GASTROENTEROLOGY

Dr. Donovan C. Browne, New Orleans

GENERAL PRACTICE
Dr. J. C. Sanders, Shreveport
GYNECOLOGY
Dr. C. H. Tyrone, New Orleans
HEART
Dr. G. E. Burch, Jr., New Orleans
MEDICINE
Dr. Edgar Hull, New Orleans
NEUROPSYCHIATRY
Dr. Gene Usdin, New Orleans
OBSTETRICS
Dr. M. A. Schudmak, Baton Rouge
ORTHOPEDICS
Dr. Rufus H. Alldredge, New Orleans
PEDIATRICS
Dr. Conway Magee, Lake Charles
PUBLIC HEALTH
Dr. R. L. Simmons, New Orleans
RADIOLOGY
Dr. George Woolhandler, Shreveport
SURGERY
Dr. C. E. Boyd, Shreveport
UROLOGY
Dr. Eugene C. St. Martin, Shreveport

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Independence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

The twentieth annual meeting of The New Orleans Graduate Medical Assembly will be held March 11, 12, 13 and 14, headquarters at the Municipal Auditorium.

Eighteen outstanding guest speakers will participate and their presentations will be of interest to both specialists and general practitioners. The program will include fifty-four informative discussions on many topics of current medical interest, in addition to clinicopathologic conferences, symposia, scientific exhibits, medical motion pictures, round-table luncheons and technical exhibits.

The Assembly has planned another interesting postclinical tour to follow the 1957 meeting in New Orleans. On Saturday, March 16, a party composed of doctors and their families, will leave from New York for the Mediterranean and Europe via plane. The itinerary includes France, Greece, Turkey, Egypt, Jerusalem, Lebanon, Syria and Italy. Arrangements have been made for medical programs in the places visited. The official tour terminates in Rome on Friday, April 12, but arrangements may be made for independent travel to any points in Europe desired, with return to the United States by air or steamer.

Details of the New Orleans meeting and the postclinical tour are available at the office of the Assembly, Room 103, 1430 Tulane Avenue, New Orleans 12, Louisiana.

HEART-LUNG MACHINE USED SUCCESSFULLY AT CHARITY HOSPITAL

The heart-lung machine was used successfully at Charity Hospital in New Orleans on August 31 in what was probably its first use on a human in the state.

The patient, a 22-year-old male, was stabbed in the left anterior chest on July 31, 1956. He was brought to the hospital in shock and an emergency thoracotomy was performed with suture of a perforating wound of the left upper lobe and a penetrating wound of the outflow tract of the right ventricle. In the early postoperative period a harsh systolic murmur was noted for the first time, and subsequent cardiac catheterization confirmed the clinical impression of a ventricular septal defect, probably acquired as a result of the stab wound. Because of the early untoward effects of the left to right shunt, operative closure of the defect was advised. Utilizing a bubble-type oxygenator, complete cardiac bypass was achieved and a repair of the defect carried out under direct vision.

The operation was performed by a team of surgeons from the department of surgery in the

Tulane University School of Medicine, assisted by physicians from the school's department of medicine.

The patient's condition following the operation was described by the head operating surgeon as "excellent" and the patient's recovery to date has been uneventful.

This case represents an unusual type of septal defect with respect to etiology as opposed to the large variety of defects of congenital origin, many of which are successfully being repaired utilizing similar techniques.

REVISED LIST OF A.M.A. FILMS AVAILABLE

A revised list of films available through the A.M.A. motion picture library has been prepared and copies are available upon request from Motion Pictures and Medical Television of the American Medical Association. This catalog lists 89 medical films suitable for showing to medical societies, hospital staff meetings and other scientific groups. The catalog also includes 45 health films of interest to physicians who may be called upon to speak before lay audiences such as service organizations, Parent-Teachers' Associations, etc.

AMERICAN RHINOLOGIC SOCIETY

Dr. Ralph H. Riggs of Shreveport and Dr. H. Ashton Thomas of New Orleans were presented the Golden Head Mirror Honor Award of the American Rhinologic Society "for meritorious sharing in the service of rhinology" at the second annual banquet of the American Rhinologic Society, Chicago, October 13, 1956.

The society held its second annual meeting October 9-13. About 100 specialists from all parts of the country witnessed demonstrations of the latest techniques in nose surgery presented at the Illinois Masonic Hospital.

Dr. Riggs participated in the workshop demonstrations and as president of the society presided over the annual meeting and annual dinner. He was elected to the board of directors upon his retirement as president, January 1, 1957.

CARE OF ARMY MILITARY PERSONNEL AWOL

In a number of cases physicians and hospitals have accepted for emergency treatment members of the Army who were in a status of absent without official leave (AWOL). Upon subsequent submission of vouchers for payment, the physician or hospital has had to be informed that current regulations preclude the payment from public funds for medical treatment rendered military personnel in such a status.

In an effort to inform interested persons of the means whereby payment may be authorized for emergency treatment of a person who is first seen in an AWOL status, it is believed that the proper procedure should be publicized through state journals.

Upon the acceptance by a hospital or physician of a member of the Army, immediate report should be made to the Army commander of the Area in which the civilian medical care is required, the chief of the military district of the area, the nearest Army post commander or the individual's commanding officer, giving the individual's name, serial number, organization, military address, status, nature of illness or injury and statement of the practicability of transfer of the patient to an Army or other governmental hospital. This procedure should be accomplished whether the person is absent with or without official leave in order that his parent organization may be informed of his continued absence by reason of illness or injury. A similar report should be rendered if for any reason an unconscious patient is believed to be a member of the Army. On receipt of an acknowledgement from military authorities authorizing the civilian hospital or doctor to treat the case, the charges for medical care furnished AWOL personnel subsequent to receipt of the authorization may be paid. These statements apply to practically every situation except when unauthorized medical care is furnished for a condition that is not an emergency.

Statements of account for payment may be forwarded to the individual's commanding officer, or The Surgeon, Fourth Army, Fort Sam Houston, Texas, who will transmit them to their proper destination.

CARDIAC ARREST SURVIVAL CHANCES INCREASING

Patients with sudden unexplained heart stoppage during surgery have a better chance of recovery now than they did five years ago, due to increasing use of new emergency techniques.

Drs. Bernard D. Briggs, David B. Sheldon and Henry K. Beecher, Boston, said that cardiac arrest is the major single cause of operating room deaths. Its occurrence has increased, but the chances of survival have also increased, because of immediate diagnosis and treatment.

The rise in the number of cases is due to the current awareness of the problem which leads to more frequent diagnosis, and to the larger number of operations performed on aged or very ill patients, they said in the April 28 Journal of the American Medical Association.

Treatment of cardiac arrest during surgery consists of prompt opening of the chest wall followed by hand massage of the heart, artificial respiration with oxygen, and use of drugs, they said.

The physicians made a study of all cardiac arrest cases at Massachusetts General Hospital, Boston, from 1925 to 1954. There was a steady decrease in the rate of cardiac arrest for the first

20 years of the period, followed by a marked increase in the last decade, particularly in the last five years, they said.

MEDICAL MALPRACTICE INSURANCE [1]

The Malpractice Insurance and Defense Board of the Medical Society of the State of New York calls attention to the very excellent article, "What Price Medical Malpractice Insurance?," which appeared recently in **California Medicine.**[2] Its author, Dr. Joseph Sadusk, Jr., is chairman of the Medical Review and Advisory Board of the California Medical Association, which has been established as a committee of the California Medical Association "to make studies and recommendations toward solution of the growing problems of professional liability insurance and malpractice actions in California."

The problem is with us whether we like it or not. It cannot be ignored or wished away. But it can be studied intelligently with the purpose of developing all the facts first and then endeavoring to arrive at sound conclusions from them.

As **California Medicine** says, editorially, "The medical profession . . . cannot withdraw from the problem of professional liability. Physicians will have to continue to live with it. Medical malpractice is therefore a problem of medicine. It is not something the profession can leave to the insurance companies. It demands the interest and cooperation of every physician. It demands the study and effective, intelligent action of every medical organization,"

[1] Editorial: New York State Journal of Medicine, Vol. 56, No. 10: 1580 May 15, 1956.
[2] California Med. 83: 389 (Nov.) 1955.

CHEMICAL TREATMENT OVERCOMES PARKINSONISM SYMPTOMS

A new proceduce of injecting chemicals into one part of the brain has been successful in restoring some "shaking palsy" victims to more normal life, three New York neurosurgeons reported recently.

The new method, called chemopallidectomy, was used on 125 Parkinsonism patients, with good results in 70 per cent of the cases, Drs. Irving S. Cooper, Nicholas Poloukhine and Aldo Morello said in the April 28 Journal of the American Medical Association.

The procedure relieves the uncontrollable tremor and muscle rigidity of Parkinson's disease (also called "shaking palsy") and related disorders without impairing the ability to move. Some patients, who had been completely or nearly helpless, could walk, care for themselves, and even work after treatment.

Chemopallidectomy consists of destroying a region in the globus pallidus, which lies in front of the thalamus. When the globus pallidus is diseased, it contributes actively to the development of tremor and rigidity. Procaine, injected into the area through a tube inserted into the brain, immediately relieves tremor and rigidity in the limbs. Later, alcohol is injected into the globus pallidus to complete the operation.

Of the first 50 patients undergoing chemopallidectomy one and a half to two and a half years ago, 65 per cent had good lasting results. Four procedures were unsuccessful, with two resulting in death.

MYOCARDIAL INFARCTION RISES AMONG WOMEN

The rate of myocardial infarction, a type of heart disease, rose sharply among women about 1940, according to a study of autopsies performed over a 45-year period in a large Midwestern hospital.

From 1910 to 1939 twice as many men as women had myocardial infarction, but from 1940 to 1954 the number was almost the same—1.1 men to 1 woman, the autopsy reports of Barnes General Hospital, St. Louis, showed.

If the ratio has actually changed in the general population since 1940 as it has in the Barnes Hospital autopsy series, it is "of profound significance," Drs. Kyu Taik Lee and Wilbur A. Thomas said in the April Archives of Internal Medicine, published by the American Medical Association.

It has been widely believed that myocardial infarction, a condition in which heart muscle cells become deadened, occurs much more frequently among men than women. The St. Louis figures contrast sharply with ratios such as 3 to 1 or 7 to 1 reported in other studies based on diagnostic information, they said. Even in Barnes General Hospital, the ratio was 2.4 men to 1 woman among patients diagnosed on admission to the hospital.

FURTHER REASON FOR THOROUGH COOKING OF PORK GIVEN

Findings of a recent study by two Yale University investigators have given further reason for making sure that pork is well cooked.

The study gave evidence that another disease, besides trichinosis, may be acquired by eating undercooked pork. The disease, toxoplasmosis, resembles pneumonia in its symptoms.

It was studied by David Weinmann, M. D., and Anne H. Chandler, M. T., New Haven, Conn. Their report appears in the May 19 Journal of the American Medical Association.

The researchers did not prove that eating undercooked pork is the only way of transmitting the Toxoplasma, but they felt that they had accumulated "very suggestive" evidence incriminating it as one transmitting agent.

The disease, caused by the protozoan Toxoplasma, is now "extremely common," they said, although 10 years ago it was considered to be

rare. In fact, in some parts of the country from 30 to 70 per cent of the population in the 40 to 60 age group is infected, they said. Normally the disease is not severe and only acute cases receive much attention.

ABSORBABLE SPONGE

The United States Patient Office has issued a patent on an artificial sponge, having the general characteristics of a conventional sponge, but which is absorbable and readily assimilable by living animal bodies.

The patent, No. 2,731,015, was assigned to the Commonwealth Engineering Co. of Ohio, Dayton, Ohio, a firm which has been a leader in the development of uses for dextran, the basic material of the new sponge.

"The very act of removing a conventional sponge from the body (during surgery) often initiates hemorrhaging," the patent states. "On the other hand, real damage results if the non-absorbable materials are not removed.

"One object of this invention is to eliminate these and other problems by providing a new porous or sponge-like material . . . which is absorbable and readily assimilated by the human body without harmful effects so that if it is placed in an incision, for example, the latter may be closed without removal of the porous material."

STUTTERING CALLED PERSONALITY DEFECT

Stuttering is a personality defect rather than a speech defect, and treatment must be aimed at the underlying emotional difficulties, reports a New York psychiatrist.

In a signed editorial in the April 28 Journal of the American Medical Association, Dr. Smiley Blanton of the American Foundation of Religion and Psychiatry said there are three functions of speech: expressing emotions, adjusting to other people, and expressing ideas.

Stuttering is a blocking of a person's ability to adjust to other people, arising from anxiety in meeting various social situations. Stutterers usually can talk to themselves, to animals, and often to people with whom they are friendly, but not in situations where fear and anxiety are aroused, he said.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

WOMAN'S AUXILIARY TO THE SOUTHERN MEDICAL ASSOCIATION

The annual meeting of the Woman's Auxiliary to the Southern Medical Association will be held concurrently with the Southern Medical Association convention at the Mayflower Hotel in Washington, D. C., November 12-15, 1956.

The program includes the usual business sessions and the special activities, which speak eloquently of Southern hospitality, include a tea, a Doctor's Day Awards Luncheon and tours of Washington, Woodlawn and Mt. Vernon.

The Southern Auxiliary originated in New Orleans, La., in 1923. Three members of the Woman's Auxiliary to the Louisiana State Medical Society have been president of Southern: Mrs. Arthur A. Herold, Mrs. Wiley R. Buffington, and Mrs. George A. Feldner. All three of these members are tireless and ardent workers in the medical auxiliaries. Mrs. Feldner designed the plaque which was presented by the past presidents, the president and the president-elect of the Auxiliary to the Southern Medical Association for their headquarters in Chattanooga, Tenn., on Oct. 2, 1956, for their Golden Anniversary Celebration. In addition, Southern further has honored one of our members by inviting Mrs. Edwin R. Guidry, Southern Councilor for La., to give the response to the welcome at the convention in Washington.

All of us who attended the State Convention in Alexandria remember Mrs. John H. O'Connell, president, Woman's Auxiliary to the Southern Medical Association, who reported on the activities of Southern. Mrs. O. W. Robinson, president-elect, will be remembered by many of us when she attended the Graduate Medical Assembly in New Orleans last March. A successful meeting is the result of careful and thoughtful planning and with Mrs. O'Connell and Mrs. Robinson in charge those attending will reap the benefits of an entertaining convention.

Mildred G. Carter, Chairman
Press and Publicity

ORLEANS PARISH

The Woman's Auxiliary to the Orleans Parish Medical Society held its first meeting of the fall season October 10, at 2:30 p. m. at the Orleans Club. Mrs. Brunswick Deutsch gave a dramatic reading.

Receiving were Mrs. Abe Golden, president of the Auxiliary and the guests of honor, Mrs. W. A. K. Seale, president of the Louisiana State Medical Auxiliary, and the past presidents of the Woman's Auxiliary to the Orleans Parish Medical Society: Mms. S. M. Blackshear, Isidore Cohn, W. Rogers Brewster, Jules Myron Davidson, Aynaud F. Hebert, Paul G. Lacroix, John S. Dunn, H. Theodore Simon, Carrol F. Gelbke, Joseph D'Antoni, Anees Mogabgab, Boni J. deLaureal, John T. Sanders, Louis Leggio, Edwin R. Guidry, Spencer B. McNair, and Robert Rougelot.

Alternating at the tea and coffee services were Mms. S. M. Blackshear, Isidore Cohn, W.

Rogers Brewster and Jules Myron Davidson.

Hostesses for the event were Mms. Lucien Alexander, Branch Aymond, Emile A. Bertucci, Sr., Richard L. Buck, John W. Bick, Jr., David Bradley, James Treadway, Fred O. Brumfield, O. R. Depp, E. H. Countiss, John C. Dubret, Frank L. Faust, T. C. Sherwood, and Gilbert Tomskey.

At the meeting it was announced that one of the Auxiliary's goals for the year is the sponsoring of another nurse through nursing school.

Mrs. Rufus Alldredge and Mrs. A. O. Goldsmith, Membership Chairmen, have added 51 new members to the Auxiliary's membership list. They made a plea to the membership to invite former members to once again become active, and to interest potential new members to join. Contact Mrs. Alldredge (Tw 5-5353) or Mrs. Goldsmith (Un 6-8741) for membership invitations.

Mrs. Jacques Magne, Tuberculosis Chairman, (Un 6-4609) asked for volunteers on Mondays to help with the Tuberculosis Seal Drive.

Kits for the Essay Contest have been distributed to schools throughout the city by Mrs. Frank Oser, Jr. and Mrs. James L. Treadway, Essay Contest Chairmen. They hope that with a choice of subjects (in the past there has only been one) even more schools and students will take an active part. The themes this year will be "The Advantages of Private Medical Care" and "The Advantages of the American Free Enterprise System".

The Auxiliary's next meeting will be held on Nov. 14.

Beryl Kelleher (Mrs. Robert C.)
Publicity Chairman
Un 6-6982

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BOOK REVIEWS

Basic Surgical Skills. A Manual with Appropriate Exercises; by Robert Tauber, M. D., F. A. C. S., Philadelphia, Pa., W. B. Saunders Company, 1955, Pp. 75, Price $3.75.

Dr. Tauber has attempted to provide a manual to instruct the novice in surgery in the basic knots and sutures that are the "ABC's" of surgical technique. He also has designed a simple training board on which these knots and ligatures can be practiced. Unfortunately, the most difficult way to learn knot-tying and suturing is through the aid of a manual. Many manuals have been designed, and I find that I, for one, usually get hopelessly lost in attempting to follow the diagrams about one-third of the way through the procedure. Consequently, my feeling is that the student can learn knot-tying with a much more economical expense of time by securing the instruction of one of his teachers for a few minutes rather than by trying to figure it out with a manual. One point of interest that might be mentioned here, is that the author recommends the use of a knot-holder instrument for tying square knots under tension. My feeling is that if the tension on a suture is such that it cannot be tied with ordinary technique, that it will not be very long before the suture will cut through the tissue or break under the strain and, accordingly, repair should be designed in some other fashion.

The instructions and demonstrations of the various basic sutures are more clear and perhaps this is the most useful part of this manual. I think that it would be of considerable value if the author had indicated where such sutures were used and for what purpose.

Another criticism that I have of this manual is that the author has devoted almost one-quarter of the book to certain techniques that he finds useful in gynecological surgery. These techniques relate to massive ligation of tissue in order to acquire hemostasis, whic I feel should be resorted to only under exceptional circumstances rather than as a "basic surgical skill."

I think, also, that there are some obvious deficiencies in description of suturing techniques and ligatures that have been neglected in this manual, for example, some of the suturing techniques employed in repair of tendons. In addition, certain basic techniques which are frequently employed in orthopedic or plastic surgery are not included.

In view of the criticisms mentioned above, I would hesitate to recommend the purchase of this manual to medical students or junior house officers.

EDWARD T. KREMENTZ, M. D.

PUBLICATIONS RECEIVED

Philosophical Library, N. Y.: Dictionary of Poisons, by Ibert Mellan and Eleanor Mellan.

W. B. Saunders Co., Phila.: Roentgen Signs in Clinical Diagnosis, by Isadore Meschan, M. D., with the assistance of R. M. F. Farrer—Meschan, M. B., B. S.; Dermatology, by Donald M. Pillsbury, M. D., Walter B. Shelley, M. D., and Albert M. Kligman, M. D.

Charles C Thomas, Publisher, Springfield, Ill.: Etiologic Factors in Renal Lithiasis, compiled and edited by Arthur J. Butt, M. D.; Urology and Industry, by Leonard Paul Wershub, M. D.